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# ANNULAR AREAS OF PULMONARY RAREFACTION IN CHILDREN

EILEEN PHILLIPS, M. D.†
and
CHESTER A. STEWART, M. D.†
NEW ORLEANS

Two decades ago lung cysts were regarded as rare, but with the increasing use of the x-ray annular areas of pulmonary rarefaction due to cysts or cyst-like changes are being discovered with considerable frequency. The literature on the subject, which is rapidly becoming voluminous, has been reviewed in several publications.[1, 10]

Studies show that cyst-like pulmonary cavities, which are not the result of the destruction of pulmonary tissue by suppuration, caseation, or parasitic infestation may be congenital or acquired and single or multiple. They vary widely in size and they may be completely or partially filled with fluid or gas. In the latter case fluid levels are discernible on chest films.

The pulmonary cysts which are considered to be congenital in origin are present at birth but they often escape detection until later in life. They are lined with cylindrical, cuboidal or flattened epithelium unless the lining membrane has been destroyed by secondary infection. In some instances the cysts produce no symptoms, but in other cases, cough, dyspnea and recurrent cyanosis may be noted. These severe manifestations are observed particularly in young infants with cysts which are expanding due to progressive inflation contingent upon a check-valve mechanism.

The detection of pulmonary cysts usually requires roentgen examination. On the x-ray plate the outline of the cyst is spherical or oval in both the postero-anterior and the lateral views. If the cyst is filled with fluid its contour aids in differentiating it from an encapsulated empyema, a condition which is more apt to have a triangular, fusiform or irregular shape.

Large cysts which occupy almost the entire hemithorax and contain air under sufficient pressure to displace the mediastinum may be confused with a tension pneumothorax. The absence, however, of a shadow of the collapsed lung in the region of the pulmonary hilum aids in ruling out the latter condition.

Like other cysts with an epithelial lining, the congenital cysts do not disappear spontaneously. For recovery their removal is required. Since there is often no definite line of cleavage around the cyst, lobectomy usually is the procedure of choice. If possible the operation probably should be deferred until the patient is two or more years of age.

An example of a congenital lung cyst seems to be provided by the first case reported in this paper. The patient, a slender five year old white female was referred to Charity Hospital for diagnosis and treatment owing to the presence of a large annular area of decreased density discovered in the upper lobe of the left lung as the result of a routine x-ray examination made prior to tonsillectomy (fig. 1). The removal of the tonsils was being considered in this instance on account of the patient's

†From the Department of Pediatrics, Louisiana State University School of Medicine and Charity Hospital of New Orleans.

poor appetite and moderate undernutrition. Although the cyst was quite large it had not caused cyanosis, dyspnea, hemoptysis, chronic cough or any other symptom suggesting its presence. Since there was no

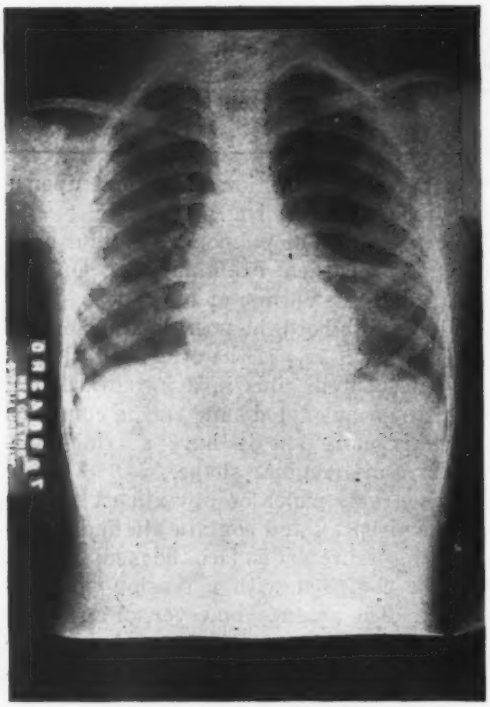

Figure 1. Congenital cyst of upper lobe of left lung.

antecedent history of severe respiratory infection or any other factor to which the area of rarefaction could be attributed, the cyst was considered to be congenital in origin.

The examination of the child's blood and urine revealed no abnormality, and the Wassermann and Mantoux tests were negative. The abnormal physical findings were limited to the thorax. On deep inspiration the expansion of the chest was symmetrical. Percussion disclosed distinct hyperresonance over the left upper hemithorax and the breath sounds were diminished in this area.

Six weeks after the patient's admission to the hospital a unilocular air-containing cyst measuring 4 x 4 x 1 cm. was enucleated under endotracheal anesthesia and the child made an uneventful recovery. At the time of operation no communication between the cyst and ramifications of the bronchial tree could be demonstrated. On microscopic examination the wall of the cyst was found to be composed of fibrous tissue lined with a single layer of nonciliated cuboidal epithelium. These observations and the absence of any history of pulmonary infection support the view the cyst probably was congenital in origin.

Cystic bronchiectasis is a second condition which some investigators regard as one form of congenital cystic disease of the lung. It is known, however, that cystic bronchiectasis may be secondary to chronic bronchial obstruction, and perhaps the majority of the cases owe their origin to this cause. These cystic dilatations are very susceptible to repeated infections which in turn favor progressive extension of structural changes in the lungs. Since cystic bronchiectasis does not respond to medical therapy the preferred treatment is lobectomy, lobulectomy, or pneumonectomy, depending on the extent of the involved area.

An example of cystic bronchiectasis secondary to chronic bronchial obstruction is provided by the second patient reported in this paper. This two year old colored female was admitted to Charity Hospital on account of fever, shortness of breath and a generalized edema of several days' duration. One month prior to admission this patient developed a respiratory infection which seemed to respond temporarily to one of the sulfonamides. According to the mother's statement the patient had been subject to repeated respiratory infections but there was no antecedent history of pneumonia, foreign body aspiration, dypnea or edema.

At the time of hospitalization the patient's rectal temperature was 102° F. and she was breathing with difficulty at the rate of 56 respirations per minute. A marked limitation of the expansion of the left half of the chest was observed during inspiration, and the percussion note was

dull over the entire left hemithorax. In addition the breath sounds were faint or absent over this area.

The chest plate revealed a density throughout the lower three-fourths of the left hemithorax. This finding combined with the pronounced lateral deviation of the trachea, mediastinum and heart indicated the presence of an extensive empyema in the left pleural cavity. There was definite evidence of fluid in the abdominal cavity and the margin of the liver was palpable five centimeters below the costal arch. In addition a generalized anasarca was present.

The examination of the blood revealed a severe anemia and a leukocytosis of 26,000 with a relative increase in neutrophils. The sickling preparation was negative and the blood urea nitrogen was normal. The serum protein value was 5.1 grams with albumin and globulin levels of 2.4 and 2.7 grams, respectively. An abundance of albumin was present in the urine. On the basis of the foregoing findings a tentative diagnosis of post-pneumonic empyema complicated by anemia, hypoproteinemia and cardiac decompensation was made.

A diagnostic thoracentesis yielded a thick purulent fluid with packed cells, fibrin and other debris constituting more than 90 per cent of its volume. Gram positive cocci were demonstrated in this fluid but their identity was not established.

The initial therapy included the administration of sulfadiazine, blood transfusions, repeated thoracenteses and digitalization with cedilanid administered intravenously. A closed thoracotomy was performed on the patient's sixth day of hospitalization but since this procedure did not provide adequate drainage an open thoracotomy was resorted to. At the conclusion of two months of therapy the child's condition was greatly improved and the gradual subsidence of the empyema disclosed the presence of several annular areas of rarefaction in the upper left lung field. In addition the presence of a bronchial fistula was demonstrated.

Owing to the persistence not only of drainage from the thoracotomy incision but also of the cyst-like areas in the lung and the bronchial fistula a pneumonectomy was performed seven and a half months after the child's admission to the hospital. The lung weighed 165 grams. The pleura was greatly thickened and a vacity attributed to an old empyema pocket was found in the inter-lobar fissure. The lower portion of the lung was composed of a dense fibrous atelectatic mass, whereas numerous cysts, the largest measuring 4 cm. in diameter, occupied the upper portion of the lung. Many of these cysts communicated with bronchi. They were lined with bronchial epithelium resting on connective tissue in which some evidence of inflammation was noted. On the basis of these findings the child's illness was attributed to an infection superimposed on congenital cystic disease of the lung. However, a few days later, when the lung specimen was being prepared for mounting, a small bean, lodged in connective tissue, was discovered in the upper left bronchus (fig. 2). In view of this finding the child's pulmonary disease was finally attributed to infection superimposed upon an acquired cystic bronchiectasis, rather than upon congenital lung cysts. This case illustrates the variety of serious complications which may accompany infection superimposed on a cystic bronchiectasis caused by foreign body as well as the difficulties which may attend the correct identification of the etiology of pulmonary changes which have the appearance of cysts on x-ray plates. After a slow convalescence the child made an excellent recovery.

Non-tuberculous pulmonary cavities in children may be due not only to evacuation of lung abscesses but also to partial bronchial obstructions which result in the production of pneumatoceles involving anatomically related segments of the lung. As a rule cavities produced by lung abscesses diminish in size as the local inflammation subsides. Occasionally, however, they persist owing either to the extensive fibrosis of the adjacent pulmonary tissue or to par-

tial or complete epithelization of the lining of the abscess cavity. Under these circumstances surgical intervention may be required particularly in instances where chronic inflammation and the development of bronchiectasis complicate the situation.

Figure 2. Cystic bronchiectasis secondary to a bean in upper left bronchus.

Pneumatoceles caused by transitory partial bronchial obstructions secondary to infections of the respiratory tract are observed with appreciable frequency in infants and young children. The pneumatoceles may attain huge size particularly in children, and occasionally display a tendency to undergo marked changes in size during the course of a few days. These cavities usually make their appearance when pulmonary infiltrations are resolving and their development is not accompanied by an abundance of purulent sputum or other signs indicative of extensive necrosis and liquefaction of pulmonary tissue. Furthermore these pneumatoceles are commonly discovered when the child is practically afebrile and is essentially free of pulmonary symptoms. Consequently by correlating clinical and serial x-ray findings areas of rarefaction due to transitory regional bronchial obstruction usually can be differentiated from chronic abscess cavities, lung cysts, and cystic bronchiectasis. This differentiation is important owing to the fact that pneumatoceles commonly disappear spontaneously in the course of a few weeks or months. Thus, they do not require the surgical measures that are needed for the cure of congenital lung cysts, chronic abscess cavities and cystic bronchiectasis.

An example of what is considered to be a pneumatocele is illustrated by observations on a three year old white female who entered Charity Hospital owing primarily to cough, dyspnea, and epigastric pain which had developed during the past few days. According to the history the child had tonsillitis three weeks prior to admission and had continued to run daily elevations in temperature. There was no other history of any previous illness. The initial laboratory studies disclosed a leukocytosis of 13,800 with a relative increase in neutrophils. The examination of the urine and the Mantoux and Wassermann tests were negative.

X-ray examination disclosed a dense shadow throughout the lower left lung field and a shift of the mediastinum to the right. On thoracentesis 40 c. c. of serosanguineous fluid was removed. Its specific gravity was 1.015 and the sediment comprised about 5 per cent of its volume. The smears and cultures revealed no organisms. Subsequent thoracenteses were unsuccessful.

The child received intramuscular injections of 80,000 units of penicillin daily. Four weeks later, when the child was convalescing and the pulmonary findings were subsiding, a large annular area of rarefaction occupying the lower half of the left lung was revealed by x-ray study. The development of this cyst-like structure was not accompanied by any exacerbation of symptoms or expectoration of purulent material. Subsequent x-rays disclosed a gradual and complete disappearance of the

empyema and pulmonary infiltration, but the area of rarefaction remained. Since the child was asymptomatic she was discharged to the out-patient department with a tentative diagnosis of pneumatocele or congenital lung cyst. Five months after leaving the hospital the x-ray plate revealed no evidence of the annular shadow. Its spontaneous disappearance provides strong evidence that the area of rarefaction was a pneumatocele.

Some of the difficulties encountered in diagnosis are also illustrated by observations on an undernourished five months old white female who was admitted to the hospital owing to a fever and cough which had persisted for three weeks following an attack of pneumonia. In this instance the urinalysis and the Mantoux and Wassermann tests were negative. A leukocytosis of 22,000 was present but there was no relative increase in neutrophils. On the basis of physical and x-ray findings the child was thought to have an unresolved pneumonia involving the lower half of the right lung. Following the administration of sulfadiazine the symptoms subsided and the pulmonary infiltration gradually resolved.

Twenty-four days after admission to the hospital and while the patient was convalescing x-ray study demonstrated a large cyst-like structure in the lower lobe of the right lung (fig. 3). The development of this cystic change was not accompanied by any exacerbations of symptoms or expectoration of purulent material suggesting the evacuation of an abscess.

Subsequently the child was discharged to the out-patient department for further observation and at the conclusion of fifteen months of follow-up study the cyst is still present but during this period the diameter of the area of rarefaction has diminished approximately 50 per cent. This change has not been accompanied by any appreciable alteration in the thickness of the distinct wall surrounding the area of rarefaction.

If further observation discloses the eventual spontaneous disappearance of this cyst-like structure it can be regarded as a pneumatocele. If, however, it persists it probably is a congenital lung cyst.

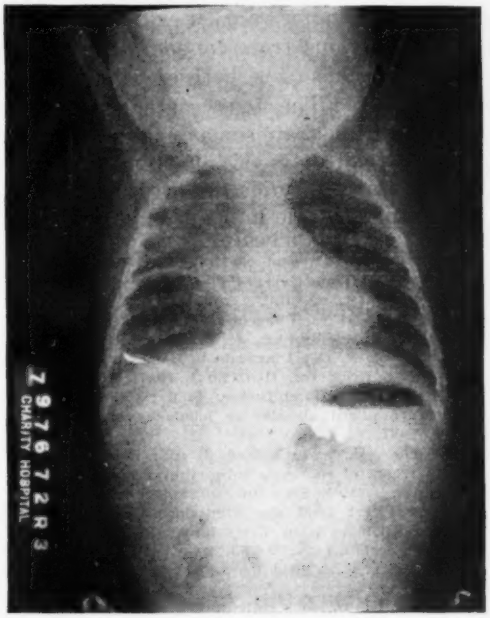


Figure 3. Pneumatocele or congenital cyst of the right lung.

## SUMMARY AND CONCLUSIONS

Cysts and cyst-like changes in the lungs of children may be congenital in origin or secondary to aspirated foreign bodies, lung abscesses, and to partial bronchial obstruction associated with infections of the respiratory tract. Cysts which show no tendency to disappear and are present in the lungs of children who have no previous history of respiratory infection or foreign body aspiration probably are congenital in origin in most instances.

Cystic bronchiectasis due to a foreign body may be confused with congenital cystic disease of the lungs. An antecedent history of foreign body aspiration is of great value in making a differential diagnosis, but unfortunately this event is not witnessed in all instances in which it occurs.

Congenital cysts and cystic bronchiectasis have several features in common. Both of these conditions may be complicated by infection and recovery requires the removal of the area of involvement in each instance.

Pneumatoceles and cavities representing

evacuated lung abscesses are inclined to disappear spontaneously in the course of weeks or months. Consequently protracted follow-up studies aid in differentiating these conditions from congenital cysts and cystic bronchiectasis, both of which persist indefinitely. The four cases reported in this paper illustrate some of the difficulties which attend diagnosis, particularly when infection accompanies the presence of cyst-like changes in the lungs of children.

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## SOME PRINCIPLES OF GENETICS

HAROLD CUMMINS, Ph.D.†
NEW ORLEANS

The branch of biology which concerns itself with the similarities and differences among individuals and the phenomena of inheritance and environment which condition these similarities and differences is known as genetics.

From our point of view, the subject matter of chief interest may be described as medical genetics because we are chiefly concerned with those phases which bear upon an understanding of the human being in health and disease. A prevalent mistaken notion about the scope of medical genetics should be corrected. To many the term suggests no more than a consideration of the genetics of disease. In truth, medical genetics is as much concerned with longevity, hardihood and resistance to disease as it is with hare lip, hemophilia, schizophrenia and a host of other manifestations of heritable diseases and susceptibilities to disease.

### HEREDITY

The fact that each type of organism—human beings, rats or flies—reproduces only its own kind is so commonplace that one is likely to give little thought to it. Human reproduction yields only human beings, rats bring forth only rats, and flies breed more flies because the reproductive cells contain inherited factors of the one species alone. Every one of us, besides possessing the traits which set apart mankind as a unique species, has traits which are distinctive of his race and family. The inherited qualities are not confined to physical features; many physiologic and psychologic traits as demonstrably inherited as eye color and hare lip.

The word congenital means "born with." An infant presenting at birth a malformation, for example, may be described as having a congenital defect, but that defect may or may not be hereditary. On the other hand, many inherited traits are not expressed at birth. It would be obviously impossible to recognize in the newborn a prospective heritable disease, or susceptibility to a disease such as rheumatic fever, which is tardy in its manifestations. As an example of an hereditary defect thus delayed in expression, atrophy of the peroneal muscles as reported by Macklin and Bowman may be mentioned. A family characterized by peroneal atrophy was traced through five generations. The onset of atrophy usually occurred within the age range of 10 to 20 years, but occasionally an affected individual did not exhibit muscular atrophy until he reached an age of about 40. In this family the trait was inherited as a dominant. Hence an individual carrying the factor for peroneal atrophy, though

†From the Department of Anatomy, School of Medicine, Tulane University.

as yet unaffected and reproducing with a normal mate, will have children of whom half are predestined to the crippling effects of this condition. Over a thousand cases of peroneal atrophy are on record in the state of North Carolina, representing more than one hundred family lines with from five to sixty affected members in each.

### ENVIRONMENT

The complex of traits present in an individual can not be wholly attributed to the action of hereditary factors. There are some characteristics—for example blood groups, eye color and a wide variety of conditions of special medical interest such as Huntington's chorea—which seem to be determined rather inflexibly by heredity. But for most traits the hereditary mechanisms are subject to influence by environmental factors, and a trait may or may not appear as anticipated because of this lability of the mechanisms which condition it.

Just what do we mean in considering environment as an agency that may cause a trait to differ from the hereditary expectation? Environment comprises, first, all conditions external to the individual: the dietary supply, temperature, light and various other radiant energies, the pressure and gaseous content of the atmosphere, relationships in the family and society—and so on. In addition, the body itself constitutes an elaborate intrinsic environment, as might be illustrated by the production of secondary sexual characters and the regulation of growth by hormonal factors. There is still more to be considered with regard to the prenatal environment of the individual. The intrauterine environment is a potent source of modification of hereditary make-up, probably especially during the earliest weeks when the most critical developmental processes are under way. Reference may be made to congenital pyloric stenosis as an example of the effects of intrauterine environment. The condition has an hereditary basis, though the mode of inheritance is uncertain. It appears likely that innate predisposition alone may not elicit stenosis and that other factors must be superimposed as triggers of the hereditary mechanism.

One indication of the effect of environmental influences in pyloric stenosis is furnished by data on primogeniture. Whatever may be the predisposing conditions associated with the initial gestation period, it is definite that the proportion of cases in first-born children is higher than would be true if birth order had no bearing on the condition. Of the 405 cases reviewed by Ford, Ross and Brown, about two-thirds were born in Toronto, over a period of 25 years. For sound comparison, the proportion of first-born children with pyloric stenosis should be contrasted with the total of all first-born in the same city. Otherwise, discrepancies would be introduced by the trend toward larger families in rural sections of the province. The only available figures on the frequency of first-born children in Toronto were for three years (1928, 1933, 1937); for these years the average value (referred to the total of 11,497 births) was 41.2 per cent—to be compared with 51.8 per cent in the series of 278 Toronto-born patients with stenosis. The difference, 10.2 per cent, was shown to be statistically significant. Moreover, the data for other orders of birth showed no indication of a relationship between birth order and the incidence of pyloric stenosis.

Another item of evidence for the conclusion that environmental factors are important in etiology is the discrepancy between the frequencies of pyloric stenosis in twins and in the single-born. In one series of 1,000 infants with pyloric stenosis (Sheldon) and in another numbering 436 infants (Ford, Brown and McCreary) the proportion of twins to single births was about twice the expected number. The state of twinning is thus shown to introduce a definite hazard.

The correlation between the conditions of a twin pregnancy and the production of stenosis has its parallel in some other traits which depend upon an environment-sensitive hereditary determination. Handedness is a good example. Left-handedness is much more frequent in twins than in single-born individuals. There is presumably a variation in the strength of the hereditary con-

trol of handedness, and the individuals in whom the hereditary determination is not firmly fixed may suffer reversal from the prospective handedness, this being almost always from right-handedness to left-handedness.

Another striking proof of disturbed environmental relationships is supplied by observations on joined twins, popularly known as "Siamese twins." Here are two individuals with structural incompletenesses which vary according to the location and extent of their union and in whom hereditary prospects are always modified by environmental factors peculiar to their development as joined bodies. A pair of Siamese twins is produced from a single fertilized ovum, hence the hereditary composition is equivalent in the two members. However, in a careful examination many differences are noted between them. In physical features and in temperament the two components are more dissimilar than are most single-ovum twins. Among the common differences is the existence in one member of situs inversus viscerum, a mirrored transposition of the visceral relationships. Such transposition may result from alteration of the purely mechanical factors which condition the visceral asymmetries. We have in our embryological collection a 12 mm. pair in which the embryos are joined front to front. In this specimen it is clear that the reversed asymmetry of heart and gastrointestinal tract lends itself to this mechanical explanation; reversal of the organs in one member is at least the most effectual accommodation to the cramped quarters in which the organs of the two embryos are developing. In Siamese twins generally there are many other differences, involving remotely related parts that do not lend themselves to so simple an explanation, but the principle of disturbed environment holds for all.

Mention has been made of the intrinsic environment, and now it should be emphasized with regard to the intrauterine period. The embryo constructs a part of its extrinsic environment, the placenta and membranes, upon which the maintenance of its life processes depends. At the same time the embryo is engaged in building the body structures and in setting them to work. The embryo, like the child, is growing and differentiating. Prenatal growth advances at an amazingly rapid pace, producing a weight increase of about six billion times in the interval between fertilization and birth. But this growth rate, remarkable as it is, is no match for differentiation as contrasted in the embryo and child. Postnatal differentiations concern structures and functions for which the framework is already laid down prior to birth. In the main the structural foundation is completed in the first eight weeks of gestation, through a succession of developmental gradations from relatively simple structures to the more and more complex. These early weeks are critical indeed for the future welfare of the individual. Not only is the organism then especially sensitive to influences in its external environment but it is peculiarly vulnerable. The most active phases of differentiation are under way and, furthermore, interference with the normal progress of a localized region or an organ may lead secondarily to effects elsewhere, so closely interrelated are the developmental processes.

From these remarks on the embryo it should be apparent that agencies of the environment, both external to the embryo and within it, may either favor the embryo or place it under handicaps. Disfavoring influences are capable of modifying the hereditary prospects of structure and function and sometimes they even lead to death of the embryo.

There remains still another important segment of environment, and that is the environment of the gametes, the sperms and egg cells in the parent bodies. These cells, prior to their union in fertilization to form the fertilized egg, or zygote, are exposed in common with the non-reproductive cells (somatic cells) to the action of the external and internal environment. If the factors are extremely deleterious the gametes may be incapable of fertilization, or development may be arrested early. Development

of the individual may proceed under milder environmental handicap, but characteristics differing from the genotype may be created. Among suggested disfavoring factors are syphilis, influenza, some metallic salts, x-rays and even the changes incident to advancing age of the mother. A delay in the meeting of sperm and egg may reduce cellular vitality to the point that fertilization is impossible or, if embryonic development ensues, departures from genotype may be produced.

We must not be over-confident in interpreting the extrinsic and intrinsic environmental effects on gametes and zygotes. Observations in man are very limited. Known conditions in the hog indicate the difficulties which stand in the way of identifying specific environmental agencies. In a pregnant sow the number of embryos commonly is less than the number of corpora lutea. When the pregnancy is advanced there is no sign indicating the fate of the eggs which did not produce embryos, but in early pregnancy their product may be discovered, in the form of blighted embryos undergoing absorption. Regional uterine pathology is ruled out as an explanation of the blighting of the embryos, and it is justified to assume that all the eggs and all the sperms concerned in the pregnancy had been exposed equally to the action of the usual environmental factors. Delay in fertilization possibly was responsible, though acceptable also as a speculation is the thought that gametes and zygotes simply differ in vigor, in this respect being like the organism as a whole.

Probably the most spectacular instance of an environmental effect on germ cells is furnished by the action of x-rays. The experimental work on this subject has been carried out especially in the fruit-fly, Drosophila, and in certain plants. The results are of interest in showing not only that the hereditary mechanism is altered in such manner that the organism differs conspicuously from the parent stock but also that it is so fundamentally affected as to transmit the new traits to the progeny. In other words, new traits known as mutations are created. Some of the new traits correspond to those already known to occur sporadically under natural conditions. X-rays increase the rate of production of such mutations, which in this experimental treatment occur with a frequency about 150 times greater than in nature. Radium rays, ultra-violet light and even heat may similarly increase the mutation rate. There are few opportunities to study the results of irradiation of the human sex cells. The sterility effects of repeated exposure to x-rays are well known, and it seems therefore plausible that there might exist an intermediate step in which the cells are altered in a way comparable to that which has been demonstrated experimentally in organisms other than man. Therapeutic dosages applied in early pregnancy are certainly capable of harming the embryo, and it seems likely that there is a critical level of dosage which might alter the sex cells. It might be remembered in this connection that irradiation of Drosophila had been applied with little result before Muller succeeded, in 1927, in devising the crucial technic which yielded a high mutation rate.

### CONSTITUTION

The search for association between conditions of medical interest and other traits (whether structural, physiologic or psychologic) lies within the province of genetics, though generally we speak of it as being concerned with constitution. Draper[1] states that constitution "should connote that aggregate or herediterial characters, influenced more or less by environment, which determines the individual's reaction . . . to the stress of environment." Tucker and Lessa[2] supply a more satisfactory definition, on which the following statement is modeled. Constitution is the sum total of the structural, physiologic and psychologic characteristics of an individual, as conditioned by both hereditary factors and influences of the environment. It is true, as Draper says, that constitution determines the nature of the individual reactions to environment; but this element is not properly a part of the definition, which in the last analysis sums up to being nothing more

than the genetics of the individual. We seek to explain how the individual came to be what he is, to determine the elements in heredity and in environment which have molded him. It is a never-ending search, since constitution is in a state of constant flux. Although the hereditary component was determined when sperm and egg cell met to initiate embryonic development, the impingement of environment is continually remolding the individual through his whole life history.

The occurrence of many a trait reflects an innate resistance or a tendency to a disease, though the trait in itself may be quite innocent of complicity. Had it not been proved, who would suspect that the virus of poliomyelitis makes its inroads especially with children whose susceptible constitution is reflected in these seemingly insignificant characteristics, among others: broad face, widely spaced central incisors, even peculiarities of finger-print types?

### VALUES OF GENETICS

There are many arguments in favor of a study of genetics by medical men. The all-embracing principle that one should know as much as he can about the human organism is sound enough, but it is too general to be satisfying. A few of the more cogent values may be specifically stated.

(1). First, we may dispose of the long-range applications in mass betterment of the human race. This constructive effort is divided into two phases. Euthenics is designed improvement of the environmental faults which jeopardize normal, healthy existence. Public health and preventive medicine may be viewed as euthenics in practice, similar aims being extended beyond the medical sphere in the field of applied sociology. Eugenics is concerned with racial betterment from the standpoint of inherited traits. Certain physical and mental traits are of substantial worth to organized society, while others are undesirable. Just what characteristics would be sought for the benefit of society as a whole is a difficult problem to decide, but everyone will agree on some of them as representing an ideal. A few other traits would as surely be classed as undesirable eugenically, among them feeble-mindedness, epilepsy and disabling physical defects.

When Guyer phrased the title of his widely known book, "Being Well Born," he did not intend to suggest that being well born is to be born to a family having wealth and good social position. He was not writing about that sort of standard, nor about the mere tracing of illustrious names in genealogy. He was concerned with being well born in the biologic sense, that is, to be born of eugenically superior stock. The aim of eugenics is to promote reproduction of those individuals whose hereditary constitution promises benefit to society, and to prevent reproduction of those who will transmit deleterious traits to their offspring. Euthenics and eugenics should advance hand in hand, but there is more lag in eugenics because of social difficulties which stand in the way of progress. The small beginnings made by segregation of the eugenically unfit and by sterilization have not struck at the real heart of the matter. These efforts, which might be described as passive eugenics, must be supplemented by an active program of popular education.

So long as human nature is what it is, the final goal of eugenics may not be reached—but we can make our way slowly toward it. It is an ironic sidelight that so much has been achieved in breeding improvements of plants and animals to meet human needs—corn, and wheat, and hogs, and cattle—while so little attention has been paid in practice to the breeding of man himself. The success attained in agricultural breeding should be encouraging. The human stock could be improved from the inside if we would only apply the same laws of inheritance which have given us disease-resistant grains, grains that can withstand severe cold, cattle that are superior for beef or for milk yield, and all the other qualities which have bettered our living from the outside.

Passing from this admittedly millennial program of eugenics, several more immediate services of genetics may be cited. (The

following passages are paraphrased from Snyder.[3])

(2). Genetics may aid in establishing a diagnosis, as exemplified in the following instance. In a case of polycythemia vera, for example, the diagnosis was at first uncertain on account of atypical elements in the syndrome; it was later substantiated by genetic findings. The diagnosis was agreed upon when the father of the patient was found to have 7,000,000 red cells, 17 grams of hemoglobin per 100 c.c., and myelocytes in the circulation. It is of interest also that a grandfather of the patient displayed erythremia.

(3). Genetics is of constant service in planning preventive measures. To give a specific example, a case of hemolytic icterus may be cited. "When seen at the age of 55, the patient had been suffering for a number of years from the incapacities of the disease, which had been unrecognized as the cause of his chronic invalidism. A splenectomy was done with good results." Had splenectomy been performed earlier the patient would have been spared the long period of disability. In view of the hereditary nature of hemolytic icterus, the seven sons of the patient were examined. None of them presented marked clinical symptoms, but five showed one or more of the laboratory signs, including microcytosis, increased fragility, increase in reticulocytes and secondary anemia. The sons were accordingly urged to submit to periodic examinations, so that treatment might be instituted promptly at need.

(4). Acquaintance with genetics equips the practitioner for giving sound advice in prospective marriages and pregnancies. The necessity of warning may be illustrated by the case of a hemophilic patient, with a positive family history, who stated that his three daughters had not been informed of the nature of his affliction and that they were not to be told. Failure to supply a warning that at least half of their sons would be hemophilic would have been inexcusable. Snyder relates the history of a young man who came to him for aid in a difficulty involving the inheritance of psoriasis. Though free himself, the disease was present in his grandfather, father, several brothers and sisters, and some nieces and nephews. His wife, then pregnant, had seen the afflicted relatives, and was obsessed with the idea that her child would have psoriasis. The fear so preyed on her that she was on the verge of a psychosis. Examination of the family history revealed that psoriasis never appeared in a child unless one of the parents had the disease. When one parent was afflicted about half of the children presented psoriasis. With these indications of a dominant heredity, it was possible to assure the distraught woman that her children would not inherit psoriasis.

## THE METHODS OF GENETICS

For the analysis of basic genetic principles chief reliance must be placed upon the results of breeding plants and animals. The classic studies of Mendel, which formed the foundation for our present knowledge of heredity, were carried out mainly with peas, though he made use of other plants and of mice. Since his day investigators have worked with a wide variety of plants and animals. Such forms are well adapted to genetic research because they may be easily bred, and generation after generation may be followed in a relatively brief span of time. The fruit-fly, Drosophila, has proved itself a most useful subject of study. The fly breeds so rapidly that twenty generations may be obtained in a year. Further, the whole experiment may be controlled both as to selection of individuals for breeding and regulation of environmental conditions. Finally, observations on chromosomes may be correlated with the genetic behavior of the traits under study, thus furthering the understanding of the inheritance mechanism. It may seem a far cry from fruit flies to man, and so it is except that both obey the same fundamental laws of inheritance. Obviously, however, the known genetic behavior of white eyes in the fly, of vestigal wings, or of certain bristles will not tell us about the pattern of inheritance of a trait in man. It leads no further than the explanation of funda-

mental mechanisms, and if we are interested in the hereditary pattern of human traits it is necessary therefore actually to observe them in family material.

Many handicaps obstruct the collection of genetic data in man. The span of a generation is long and families are small, hence it is difficult to assemble extensive pedigrees for tracing the transmission of hereditary characteristics. There is of course no control of mating, such as may be readily applied in the breeding of plants and animals. Environmental circumstances also are beyond control, and indeed are even largely unknown. This latter deficiency is of great moment in many conditions of medical importance, wherein environmental factors may either exercise a trigger action on the hereditary mechanism or suppress its potential effects. To make the situation even worse for the student of human genetics, it is usually possible for him to examine personally only a small proportion of the individuals whose histories are pertinent. He is forced to record the remaining individuals as "unknown" with respect to the traits in question, else rely upon the statements of others as to whether these individuals are positive or negative. It is clearly apparent that unreliable diagnoses would seriously vitiate the record and its genetic interpretation. In spite of these and other difficulties, much has been done and much is being done with human genetics, including its medical aspects.

### THE APPROACH IN HUMAN GENETICS

Several analytic methods are available in analyzing the inheritance of traits in man.

(1). The oldest and most familiar is the pedigree method, the construction of a family tree which shows the relationships of all members of the family who are accessible for record and indicating the status of each with regard to the trait which is being studied. By inspection of such a tree one may determine quickly the number of individuals affected, the manner of transmission from parent to offspring and the distribution by sex. All these items are essential to interpretation of the mode of inheritance. Of equal importance in those conditions where the hereditary mechanism is delicately responsive to environmental factors is a search for all possible agencies that might have favored or disfavored the expression of the inherited traits. The search is beset with difficulties.

The initiation of a study of this sort is often merely the arousal of scientific curiosity in one whose chief interests lie outside the field of genetics. As a case in point, and as an exemplar that should be followed by more medical men, I shall refer to a study published by Harvey Cushing in 1916. Ten years before this publication, Cushing was consulted by a woman with a cerebral glioma. He noted that her fingers could not be bent at the proximal interphalangeal joints. An uncle who accompanied her was likewise affected, and it developed that the family physician, also with the patient, had married her fifth cousin who had the same condition. This casual observation led to an extensive study of the family line. Records of 452 persons, representing seven generations, were obtained. It was shown that this ankylosis, or symphalangy, is inherited as a simple Mendelian dominant, the offspring of affected individuals thus having a 1:1 chance of presenting the anomaly.

Before leaving the subject of family trees it must be stressed that family incidence of a trait is not always a sign that the trait is inherited. Conformity of the distribution of cases to one or another of the recognized modes of hereditary transmission is of course suggestive that a trait is inherited, but the investigator is always watchful for environmental factors that may influence the inheritance, or perhaps even be responsible for the effects observed in a family group which is subjected to the same disfavoring environment. Night-blindness may be selected in illustration. In some families the defect appears to be inherited as a dominant trait, in others as a recessive and in still others a sex-linked recessive. Such inconsistencies of genetic behavior are not unknown in some other traits, but one wonders to what degree the dietary regime may figure as the cause of this irregularity in

the mode of inheritance of night-blindness, if indeed in some family groups a deficiency might not produce a false appearance of inheritance.

(2). Consanguineous marriages may elucidate the hereditary mechanism, but only when the occurrence of the trait concerned is very rare in the general population (less than 0.1 per cent). When consanguineous marriages result in a high family incidence of traits so infrequent as this it is to be concluded that they are inherited recessively.

(3). Twins and other multiple births are a fruitful source of genetic information. Twins, in the restricted sense of a pair rather than a larger number of individuals produced in the same pregnancy, are of two main types. Dizygotic twins are the product of two zygotes, two fertilized eggs which jointly undergo their intrauterine development; such cases are appropriately termed two-egg, or dizygotic twins; sometimes they are called fraternal twins. The so-called identical, or better monozygotic, twins arise from a single fertilized egg. The production of two individuals instead of one from a single zygote results from fission of the embryo at a very early period. When separation of the two embryonic rudiments is incomplete the twins are united as "Siamese twins" or as some less fully duplicated type of double monster.

A prerequisite for reliable results in twin studies is assurance that the pairs of twins under investigation be correctly diagnosed as to zygotic origin. The usefulness of the twin material lies in the fact that the two members of a pair of monozygotic twins have an equivalent hereditary constitution, while dizygotic twins are generally like separately born siblings except that the two individuals have had more in common in their intrauterine and postnatal environments. Sound diagnostic methods are accordingly essential when twins are used in the analysis of genetic phenomena. One of the criteria of zygosity is sex, but its helpfulness is limited. If the twins are opposite-sexed the diagnosis of dizygosity is at once established, but same-sexed twins may be either monozygotic or dizygotic. In differentiating the two types of same-sexed twins special diagnostic procedures must be applied. The similarity method is based upon exacting comparisons of the two members of the pair. In certain traits unlikeness, or discordance as it is called by students of twins, would at once distinguish the pair as dizygotic. For example, if blood groups are discordant we can be as sure that the pair is dizygotic as if the pair were unlike-sexed. The mode of inheritance of blood groups, like sex, is such that two individuals arising from one zygote must be concordant. Unfortunately there are not many human traits that are so rigidly predetermined by inheritance, and unless there is discordance in such traits examination of the twins must be extended to a variety of other characteristics. The general principle of the similarity method is that twins which most closely resemble each other are monozygotic, to a high degree of probability. Twins which present larger differences are most likely dizygotic.

What can be done with twins as a source of genetic information? The possibilities of medical applications are almost unlimited. The observations will enable us to determine what traits are inherited and to evaluate the relative influences of heredity and environment. H. H. Newman, an active investigator of twins through many years, has gone so far in this test of the roles played by "nature" and "nurture" as to assemble a series of twins who were separated in infancy or childhood and raised in quite different surroundings.

Perhaps examples will supply a clearer picture of the methods employed in genetic analysis of twins. The gathering of even isolated records is profitable, as shown by the following cases. Champlin reports the development of sarcoma of the testis in both members of monozygotic twins. In the instance of one twin, sarcoma developed in the right testis at the age of 24; earlier, that testis had been traumatized. Operation was delayed, and the man died at 26. The other twin, in the absence of known trauma, developed at the age of 31 the same

type of sarcoma, and like the twin brother in the right testis. The close correspondence of the pathology in these two young men suggests at once an hereditary background for the malignancy, and there is the further suggestion that its activation in the first case was hastened by trauma. The hereditary basis of neoplasia as revealed in twins could be illustrated by enumerating many instances, similar to those here listed, of correspondence of type and location in the two members of a monzygotic pair; retinal blastoma of the left eye; uterine cancers; ovarian cysts on the same side; cerebral gliomata; malignant ovarian teratomata; adenomata located in the same quadrant of the left breast, appearing in both twins at the early age of 21.

Studies of mass samples of twins are more fruitful. Let us turn to such a study, in this instance one designed to yield a measure of the parts played by heredity and environment in schizophrenia. Much work has been done on the genetics of schizophrenia, and that there is often an hereditary basis is well recognized. This example is therefore chosen only to show the twin method in operation. Rosanoff assembled a series of twins in which one or both members presented the affliction. Among monozygotic pairs both members were schizophrenic in 68 per cent of the pairs, while in dizygotic pairs both members were schizophrenic in but 15 per cent of the cases. There is thus shown a considerable degree of lability in the action of the hereditary factors. If their effects were inevitable, schizophrenia would have occurred in 100 per cent of the monozygotic pairs instead of 68 per cent. Further, an incidence of 15 per cent of doubly affected dizygotic pairs is rather too high to be explained by an independent hereditary determination, showing that environmental experience shared by the two members of a pair must have increased the occurrence of schizophrenia beyond the hereditary expectation. The findings again bring emphasis to the interplay of heredity and environment as agents in the constitution of an individual.

Another illustration of the method, applied to a different pathologic trait, may be of interest. I have chosen Lemser's study on diabetes mellitus. This author, in addition to recording family trees which show hereditary transmission, reports on the occurrence of diabetes mellitus in 55 pairs of monozygotic twins and 79 pairs of the dizygotic type. Collection of the data on twins, as in the investigation of schizophrenia, consisted in a search for twins in which at least one member of the pair was affected. In Lemser's series 85.5 per cent of the monozygotics were concordant, that is, both members were diabetic; in the dizygotic only 29.1 per cent were concordant. Again it is evident that the conditioning of diabetes through heredity is not rigid. If diabetes were inflexibly determined by heredity 100 per cent of the monozygotic twins would be concordant. Of course, it is justified to assume that the as yet unaffected members of the discordant monozygotic pairs must be highly susceptible. Metabolic dysfunction might be readily precipitated in them by exposure to any of the factors which are of etiologic importance in diabetes mellitus.

(4). The discussion of family trees and of the special methods employed in twin studies has taken us a long way from our subject heading—namely, the approach in human genetics. We are now ready to resume the listing of the varied sources of genetic information in man. Several different sources remain unmentioned, but of these only one which is regarded as being of particular importance will be discussed. The experimental animal may be of service when the condition in question is of such nature as to admit comparison between animal and man. Thus, a large amount of evidence on the hereditary pattern of malignancy in rats and mice is available for supplementing observations on the genetics of malignancy in man. However, observations in animals can not be transferred blindly to an interpretation of human genetics, because species differences are known to exist. Cattle, for instance, differ from man in the genetics of malignancy of the mammary

gland. Nothwithstanding that the mammary is a common site of malignancy in mammals generally, including man, only two cases in the cow are on record. Considering also that the udder is maintained at a functional level through a large share of the lifetime of a cow, and that it is subjected to frequent and often severe trauma, it is clear that neither the hereditary constitution nor the commonly postulated environmental factors predispose to mammary cancers in this species.

To present an example of studies in animals, reference may be made to their use as a test of the inheritance of resistance to infectious disease. It had been shown long ago by botanists that resistance and susceptibility to plant diseases are inherited. On the premise of a unity in fundamentals among all living organisms, it might have been predicted that resistance and susceptibility in man and other mammals also are inherited. Now we know that this is true, partly from direct observations on human families and partly from experiments on laboratory animals. Webster, working with mice under carefully controlled environmental conditions, was able to demonstrate that some strains are resistant to certain virus and bacterial infections, while other strains are susceptible. There was a differential among the infections; a strain resistant to one type of infection often proved susceptible to another. Crossings of the resistant and susceptible strains indicated that resistance is inherited as a Mendelian dominant.

### CONCLUSION

This discussion is designed only to stress the indispensability of genetic principles in medical thinking. It makes no pretense at detail, since consideration of detail is impossible when the basic concepts must be covered in such brief form. The aim will have been accomplished if the following generalizations are appreciated. Factors of heredity determine conditions of health as well as some malformations, diseases and predispositions to disease. Such heredity is inflexible in certain traits, but for many traits the mechanisms of heredity are labile. Hereditarily prospective traits may be either sustained or altered by environmental influences, which are effective on the sperm and egg and on the individual from the very beginning of his existence as an embryo.

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## APPRAISAL OF THE NUTRITIONAL STATUS OF CHIDREN*

ARILD E. HANSEN, M. D.†
GALVESTON, TEXAS

Whether we have war or peace, depression or prosperity, plague or freedom from pestilence, the subject of nutrition will always be of importance to the physician. It is the purpose of this presentation to review certain practical aspects of the nutritional problem which may aid the student of medicine as well as the practicing physician in understanding the overall problem of nutrition in children.

If we were completely susceptible to propaganda and allowed our thoughts and actions to acquiesce to the repetitious statements of the radio, the artistic advertising displays in the magazines and newspapers, and the remarks of the provocative politician and the social ideologist, we, as physicians, might find ourselves deprived of one of the most important phases of the practice of medicine, namely, nutrition. True enough, the field of nutrition is not exclusively ours, as it is indeed broad and many individuals and groups, such as the soil conservationist, farmer, rancher, breeder, manufacturer, transporter, distributor, baker, cook, and retailer are concerned with the overall problem of supplying nutrients for use by the body. Many others such as

*Presented as a special lecture at the Louisiana State University School of Medicine, New Orleans, Louisiana, April 13, 1945.

†From the Department of Pediatrics, University of Texas School of Medicine, Galveston, Texas.

the chemist, physicist, biochemist, botanist, home economist, and dietitian have contributed greatly to our knowledge of nutrition. However, it is the physician who has the dual knowledge of fundamental nutrition and clinical medicine and thereby is capable of performing the important task of assessment of the nutritional state of the individual.

This problem is especially important in the appraisal of the nutritional status of children and indeed at times is very difficult. The physician, as well as many others, has expended considerable effort to find a simple, easy method for the evaluation of the nutritional status of children, but to date none such has been devised. The time honored custom of relying largely or entirely upon clinical impressions expressed as "well nourished" and "poorly nourished" is a far cry from the real appraisal of intake and utilization by the body of the specific nutrients. On the other hand, application of the exact methods used in animal studies is obviously impossible, and results obtained therefrom cannot be applied directly to human subjects. The task remains a difficult one, but it cannot be shirked by the physician, as he is the only individual equipped with the necessary knowledge and experience to make this evaluation. This information is essential to him in the care of his patients in order that he may, first, diagnose deficiency disease and institute proper treatment, and second, maintain ideal or optimal nutrition by preventive measures for those under his care.

In the modern, enlightened household, actual deficiency disease is seldom encountered; however, the physician in his examination of children frequently finds evidence of malnutrition which cannot be explained on the basis of dietary lack. It is in such situations that the physician plays his particular role, for there are a number of conditions which may be the underlying cause of this malnutrition. The condition may be that of faulty absorption of nutrients, such as found in cystic fibrosis of the pancreas and other steatorrheas; it may involve disturbances in intermediary metabolism, such as in diabetes mellitus and other endocrine disturbances and in certain liver disorders; or it may be due even to the presence of infections, such as rheumatic fever, tuberculosis, syphilis, brucellosis, or nephritis. The possibility of the presence of these or other disorders must be carefully weighed in the physician's appraisal of the nutritional status of his patient.

Although the future may bring forth a satisfactory short-cut method, the best means at his disposal at the present for this evaluation are those employed in arriving at any other diagnosis: namely, (1) complete history; (2) careful physical examination; (3) employment of laboratory procedures; (4) knowledge of the nature of the disease, in this instance, the basic aspects of nutrition. The following outlines adapting these procedures to the specific problem of nutrition may be of some assistance to the student and physician.

### FEATURES OF THE HISTORY TO BE CONSIDERED IN NUTRITIONAL EVALUATION

*Age of patient*: Most of the serious and more common nutritional deficiency states (rickets, tetany, scurvy, xeropthalmia) occur more frequently in the infant and young child than in the older child. Also, the manifestations of deficiency disease may vary with age. In vitamin A deficiency the hyperkeratosis which occurs in the older child seldom is seen in the infant, and keratomalacia is found more frequently in young infants than in older children. Symptoms of nicotinic acid deficiency are scarcely known in infants yet older children may suffer from typical pellagra. Infantile beriberi is entirely different from the thiamin deficiency picture seen in older children and adults.

*Nature of the presenting complaints*: The nature of the presenting complaint may provide clues which lead one to suspect nutritional deficiency. For example, the information that the baby is fussy and cries when touched, as in the changing of the diaper, and has a tendency to bleeding gums may suggest scurvy. A history of convulsions between the ages one to three years

may suggest tetany. The symptoms of lassitude, lack of endurance, and disinterested attitude may lead one to suspect either protein or iron deficiency. There is some tendency to list individual symptoms such as insomnia, anorexia, weight loss, lacrimation, lassitude, and many others which are said to occur in various deficiency states. Inasmuch as these same symptoms are commonly found in many pathologic psychosomatic states to list these as evidence of deficiency disease tends to confuse the inexperienced and to mislead the ill informed person. The interpretation of the significance of various complaints is possible only in the light of a complete dietary history, physical findings, certain laboratory data, and therapeutic tests.

*Family background:* The constitutional heredity of the parents as to body type and pattern of weight gain may be of inestimable value in the interpretation of the child's height and weight measurements at any particular time. The psychologic and emotional reactions of other family members are important in the interpretation of nutritional problems in children.

*Economic factor:* It has been stated that of all factors which contribute to malnutrition, the socio-economic factor is the most important. Where poverty is found, malnutrition exists.

*Dietary history:* Careful analysis of the actual food consumption by the child gives valuable information regarding the likelihood of development of a deficiency disease. Every student of medicine, for example, knows that it is risky to make the diagnosis of scurvy or rickets without the dietary history of an insufficient intake of citrus fruit or vitamin D, respectively. Knowledge of the amount of and length of breast feeding likewise is significant as scurvy, xerophthalmia, and rickets are seldom seen in infants fed on human milk. In breast fed infants information on the adequacy of the maternal diet is also essential for complete evaluation. A detailed dietary history is especially important in children with allergic disorders, food idiosyncrasies, or such diseases as diabetes, nephritis, nephrosis, and celiac syndrome. In fact, dietary restrictions may have occurred with any disease, or during the recovery state following it. A very useful method*, approved by the Council on Food and Nutrition of the American Medical Association, has been devised by the Department of Nutrition of the National Live Stock and Meat Board for analyzing the food consumption of dietary

*National Live Stock and Meat Board, 407 South Dearborn, Chicago, Illinois. Nutrition Yardstick: A graphic calculator for measuring the food value and adequacy of daily diets.

TABLE 1.
RECORD OF DAILY FOOD CONSUMPTION FOR EVALUATION OF THE DIETARY HISTORY OF THE CHILD

| Type of food | Daily Consumption | | Allowance for good nutrition for growing child |
|---|---|---|---|
| I. Milk and milk products | Total | | 1½ pts. (½ - 1 qt.) daily |
| | With meals | Between meals | |
| | With cereal | In cooking | |
| | Cheese | Cream | |
| | Others | | |
| II. Eggs | Total | | 1 serving daily or 5 times per wk. |
| | With meals | In cooking | |
| III. Meats | Total | | 1 serving daily or 5 times per wk. |
| a. Lean (muscle) | Beef | Pork | |
| | Lamb | Others | |
| b. Poultry | Chicken | Others | |
| c. Fish | Salmon | Others | |
| d. Shell fish, etc. | Shrimp | Crab | |
| | Others | | |
| e. Liver | | | Once per week. |
| IV. Vegetables | | | |
| a. Potatoes | Total | | 1 serving daily |
| | Irish | Sweet | |
| b. Leafy green | Total | | 1 serving daily |

| Type of food | Daily Consumption | | | Allowance for good nutrition for growing child |
|---|---|---|---|---|
| | Lettuce | Chard | | |
| | Broccoli | Brussel sprouts | | |
| | Cabbage | Others | | |
| c. Others | Total | | | 1 serving daily |
| | Beans | Peas | Corn | |
| | Asparagus | Beets | | |
| | Carrots | Cauliflower | | |
| | Parsnips | Squash | Turnips | |
| V. Fruits | | | | |
| a. Citrus and tomato | Total | | | 1 serving daily |
| | Orange | Lemon | | |
| | Grapefruit | Tomato | | |
| b. Others | Total | | | 1 serving daily |
| | Peaches | Apricots | | |
| | Pears | Apples | | |
| | Figs | Pineapple | | |
| | Plums | Berries | | |
| | Bananas | Prunes | | |
| | Others | | | |
| VI. Breadstuffs and cereals | Total | | | 1 slice bread or equivalent in cereal with each meal |
| a. Bread | Total | | | |
| | Whole Wheat | Enriched | | |
| | Others | | | |
| b. Cereals | Total | | | |
| | Cooked | | | |
| | Whole grain | Enriched | | |
| | Macaroni | Spaghetti | | |
| | Soybean | | | |
| | Others | | | |
| | Prepared | | | |
| | Whole grain | Enriched | | |
| | Others | | | |
| VII. Fats | Total | | | 2 tablespoons daily |
| a. Butter or fats with vit. A | Total | | | |
| | As spread | In cooking | | |
| b. Animal fats and certain vegetable oils as a source of essential fatty acids | Total | | | |
| | Lard | Meat Fats | | |
| | Corn oil | Soybean oil | | |
| | Others | | | |
| VIII. Vitamin D Dietary supplements | Total | | | Equivalent in vitamin D content to teaspoon of cod liver oil |
| | Cod Liver oil | Concentrate | | |
| | Percomorphum oil | Viosterol | | |
| | Irradiated milk | | | |
| IX. Iodized salt | Total | | | Trace—(if in goiter belt) |
| X. Desserts | Total | | | As need for calories |
| Sugar and syrup | Sugar | Syrup | | |
| | Jams | Jellies | | |
| | Cake | Cookies | | |
| | Ice Cream | Pudding | | |
| | Custards | Jello | | |

essentials in terms of the ordinary food servings.

The following outline (table 1) may be employed to good advantage in obtaining the dietary history of the child.

After a complete dietary history has been obtained the student often is at a loss, other than for citrus fruits, vitamin D and iodine, to know how to interpret the findings in relation to the occurrence of deficiency disease. To facilitate the interpretation of the dietary history we have presented schematically in table 2 the specific deficiency states which may be suspected if dietary analysis discloses an insufficiency in any or several of the specific food categories. Study of the chart will also show the food classes which supplement each other in providing the various food essentials.

TABLE 2.
DEFICIENCY DISEASES WHICH MAY BE SUSPECTED WHEN DIETARY HISTORY REVEALS AN INSUFFICIENT INTAKE OF FOOD IN THE VARIOUS CLASSES

| | I MILK | II EGGS | III MEAT | IV VEG. LEAFY | V FRUITS CITRUS | VI CEREALS WHOLE | VII FATS VIT. A | VII FATS ESS. F.A. | VIII VIT. D | IX IODINE | X DESSERTS |
|---|---|---|---|---|---|---|---|---|---|---|---|
| Edema | xx | x | xxx | | | x | | | | | |
| Anemia | | x | xx | x | | x | | | | | |
| Rickets<br>Tetany<br>Osteoporosis<br>Caries ? | xxx | | | x | | | | | xxx | | |
| Xerophthalmia<br>Nyctalopia<br>Phrynoderma | xx | x | | x | x | | x | | | | |
| Scurvy | | | | xx | xxx | | | | | | |
| Beriberi | | | xx | x | | x | | | | | |
| Pellagra | x | | xx | | | x | | | | | |
| Ariboflavinosis | xx | | x | x | | x | | | | | |
| Dermatoses | | | | | | | | ?x | | | |
| Goiter | | | | | | | | | | xxx | |
| Underweight | x | | x | | | x | | | | | xx |

THE PHYSICAL EXAMINATION IN NUTRITIONAL EVALUATION

Examination of the child with the knowledge of the complete dietary history at hand aids greatly both in the detection of distinct deficiency disease and in the consideration of findings which may or may not be due to specific lack of food substances. This, of course, is not possible if the examiner is not familiar with the manifestations of the various deficiency diseases. Oftentimes, however, the interpretation of such findings as lacrimation, photophobia, and burning of the eyes; sores at angle of mouth; skin eruptions; bleeding gums; glossitis; weakness or prostration; edema; paralysis; convulsions; aphonia; pallor; underweight, and others may be difficult without the support of the history and the knowledge of response to specific therapeutic measures. Conversely, if the physician finds evidence of deficiency states in spite of an apparently adequate dietary history then he must leave no stones unturned to determine the cause; for example, certain disease conditions or deranged metabolic states. In general, of distinct value to the physician is the clinical impression he gains regarding the patient's appearance, color, muscle tone, tissue turgor, mental alertness, and physical activity, all of which give him valuable information. This impression must be interpreted in the light of other data, but by and large, it is safe to say that a child radiating health and vigor rarely has a deficiency disease.

Height and weight indices have fallen somewhat into disrepute as a guide to nutrition, but those giving the normal range are still of value. The Grid Method* for evaluating physical fitness as devised by Wetzel has been found by some to give valuable information. The child making a satisfactory gain in weight seldom is suffering from a deficiency disease.

LABORATORY DATA USEFUL IN EVALUATION OF NUTRITIONAL STATUS

In table 3 are listed laboratory procedures which are of assistance in the evaluation of the nutritional status. In children the biomicroscopy or slit lamp studies are not too satisfactory as it is difficult to obtain the necessary cooperation especially from the small child.

*Prepared by NEA Service, Inc., 1200 W. 3rd St., Cleveland, Ohio.

TABLE 3.
SUMMARY OF LABORATORY PROCEDURES WHICH MAY BE USED IN THE DIAGNOSIS OF VARIOUS DIETARY DEFICIENCY DISORDERS

| CONDITION | X-RAY | BLOOD | URINE | BIOMICROSCOPY | OTHER | |
|---|---|---|---|---|---|---|
| Rickets<br>Tetany<br>Osteoporosis | Long bones<br><br>Skeleton | Serum Ca, P, phosphatase | | | | |
| Edema | | Plasma protein | Creatinine output | | | |
| Scurvy | Long bones | Ascorbic acid Saturation test | Excretion, saturation | Gums | Tourniquet test | |
| Xerophthalmia | | | | | Corneal smear | Dark adaptation test |
| Keratomalacia<br>Nyctalopia | | Vitamin A | | Conjunctiva | | |
| Phrynoderma | | Carotene | | | Skin biopsy | |
| Anemia | | Hb, Rbc, hematocrit | | | Bone Marrow | |
| Beriberi<br>Pellagra<br>Ariboflavinosis | G-I tract<br>Heart | Pyruvic acid thiamine | (Pyrimidine) Excretion Saturation | <br>Tongue<br>Cornea | EKG | |
| Hemorrhagic disease | | Prothrombin time | | | | |
| Certain dermatoses | | Iodine number of serum fatty acids | | | | |

OUTLINE OF BASIC FACTORS IN NUTRITION

As an aid for evaluating whether the findings obtained from the history and examination are indicative of adequate nutrition, the approximate daily requirements for basic factors are summarized in tables 4, 5 and 6.

I. *Water:* Next to oxygen, water is the most important substance needed by the body to maintain normal metabolic functions. The infant's need for water is approximately three times that of the adult subject in relation to body weight. The approximate water requirements for various ages are presented in table 4.

TABLE 4.
APPROXIMATE DAILY REQUIREMENTS OF CHILDREN FOR CALORIES, PROTEIN AND WATER

| Age yr. | Calories per kg. | Protein gm./kg. | Water c.c./kg. |
|---|---|---|---|
| Infancy | 110 (120 - 100) | 4.0 | 150 |
| 1 - 3 | 100 (100 - 90) | 3.5 | 125 |
| 4 - 6 | 90 ( 90 - 80) | 3.0 | 100 |
| 7 - 9 | 80 ( 80 - 70) | 2.5 | 75 |
| 10 - 12 | 70 ( 70 - 60) | 2.0 | 75 |
| 13 - 15 | 60 ( 60 - 50) | 1.5 | 50 |
| 15+ | 50 ( 50 - 40) | 1.0 | 50 |
| Adult | 40 ( 40 - 30) | 1.0 | 50 |

II. *Calories:* In relation to body weight the energy needs of infants are about three times that of adults. The approximate caloric requirements for the various ages are given in table 4. In the average diet about 50 per cent of the calories are supplied by carbohydrates, 35 per cent by fat, and 15 per cent by proteins. The greatest variation in expenditure of calories occurs in regard to growth and activity, the basal requirements are proportionally the greatest and are quantitatively more in infants than older subjects, while those necessary for specific dynamic action are small but quite constant and the unused portion in the stool usually does not amount to more than 10 per cent of that consumed. In the consideration of caloric requirements in practice, the measurement of success in meeting the caloric needs of the individual is based upon such clinical data as growth, general well being of the child, and satiety.

III. *Proteins:* Proteins are the predominant solid constituent of the body and in the diet are the only source of the essential amino acids. The ten amino acids, threonine, valine, leucine, isoleucine, lysine, histidine, trypophane, phenylalanine, methionine, and arginine which have been found to be indispensable for growth and

development of experimental animals in recent years, have been found to be the ones essential for human nutrition, with the exception of histidine. Although the biologic value of animal protein is greater than that of vegetable proteins, it is known that mixtures of these proteins as they are consumed in the usual diet supply the required amino acids. The protein needs of the infant, just as for water and calories are approximately three times greater in relation to body weight than those for adults. The approximate requirements for the various ages are presented in table 4.

IV. *Carbohydrates:* The greatest percentage of the calories and necessary bulk of the diet are supplied by carbohydrate, yet probably less than one per cent of the body weight constitutes stored carbohydrate—chiefly as glycogen. Carbohydrate calories are a readily available source of energy and sufficient quantities should be supplied in the diet to prevent the ketosis which results when excess fat is burned to supply the caloric needs. If the calories supplied by proteins constitute 30 per cent or more of the calories used, ketone bodies do not accumulate in the blood or tissues even though no carbohydrate is included in the diet. The relative quantity necessary in the diet of children is the same as for adults.

V. *Fats:* The chief dietary function of fat is to supply fatty acids in the form of triglycerides as a source of concentrated energy. The nutritional significance of the lipids or fat-like substances, however, are not entirely understood. No one has demonstrated the dietary necessity of the compound lipids, phospholipids (lecithin, cephalin, or sphingomyelin) or glysolipids (cerebrosides). Of the derived lipids, cholesterol is the most abundant of the animal sterols yet its synthesis occurs in the body. However, the body does have special need for certain sterols when irradiated as a source of vitamin D. Choline, derived from the phospholipids, is considered as one of the components of the vitamin B complex. Of special interest from the viewpoint of nutrition are the so-called essential fatty acids, the most important of which are the highly unsaturated fatty acids, linoleic acid (18 carbon atoms with 2 double bonds) and arachidonic acid (20 carbon atoms with 4 double bonds).

*Essential Fatty Acids:* Burr and Burr[1] in 1929 found that young rats reared on diets extremely low in fat developed a syndrome characterized by retarded growth, scaliness of the skin and tail, impaired reproductive capacity, kidney lesions with hematuria and early death. Administration of small amounts (20 to 100 mg.) of linoleic acid or arachidonic acid effected a complete cure in these animals. Hansen and Wiese[2] found that when puppies were fed diets practically devoid of fat (0.13 per cent), the skin and hair showed abnormalities without any evidences of disturbance in growth or kidney damage. When these animals were about three months of age the hair became dry and coarse and the skin was dry and exhibited extensive flaky desquamation. Litter mate animals which received fresh lard substituted isocalorically for sucrose calories had normal skin and hair. Blood lipid studies revealed that both rats and dogs, although they synthesized sufficient fat to maintain normal levels in the blood, did not synthesize fat with a high degree of unsaturation.

Relatively little is known of the human need for these highly unsaturated fatty acids. On several occasions human subjects have been kept on diets very low in fat without any distinct clinical syndrome developing. Blood lipid studies, however, indicate that the human organism is not able to synthesize highly unsaturated fatty acids. Even though a specific human need for these as yet has not been proved fully, there is some indirect evidence that linoleic and arachidonic acids may play a role in human nutrition. The probable function of these acids is in reference to the condition of the skin. The author has observed that certain infants with eczema appear to be benefited clinically when fats such as fresh lard and vegetable oils (corn oil and linseed) are added to the diet. Although not all patients with eczema respond favorably,

in the experience of the author and of Finnerud and co-workers[3] it appears that more than one-half of the patients with intractable eczema are materially benefited by the dietary inclusion of such fat. These fats (fresh lard and certain vegetable oils which contain the essential fatty acids) are effective in curing and preventing the fat deficiency syndrome in experimental animals. The clinical evidence with patients with eczema is further substantiated by blood lipid studies. It has been found that the serum fatty acids of the greater majority of infants with eczema and over one-half of the adult subjects with chronic eczema have iodine numbers which are below the normal range. Coincident with the amelioration of the skin manifestations in those patients given dietary fat rich in the unsaturated fatty acids the iodine numbers of the serum fatty acids in many patients return to normal levels. Much is yet to be learned concerning the metabolism of the unsaturated fatty acids. Our understanding of the problem in regard to human beings will be enhanced no doubt when satisfactory methods are devised for the determination of linoleic acid and arachidonic acid in the blood and tissues. Interrelationships have been found to exist between the essential fatty acids and certain vitamins such as pyridoxine, pantothenic acid, inositol and possibly vitamin A metabolism. Based on studies with experimental animals Burr has suggested that the diet should contain about one per cent of fat rich in the essential fatty acids.

VI. *Minerals:* Minerals is the term more or less loosely applied to all elements which are normally essential to structure and function of the body other than those which occur largely as organic components of tissue (carbon, nitrogen, hydrogen, and oxygen). The minerals occur largely as ash when a food or body tissue is burned. Inasmuch as minerals play important structural and functional roles, there exists a special need for them during the period of growth. Marriott has estimated that for each gram of protein laid down in the body 0.3 gm. of minerals also is deposited.

Electropositive elements:

*Calcium:* Essential for structure in bones and teeth, also important in metabolism of muscle nerves and coagulation. Usually an intake of 50 to 70 mg. per kilogram per day in children will provide a retention of 10 to 20 mg. per kilogram per day which amount appears to be an optimal quantity. The chief source is milk and milk products. In table 5 the approximate daily requirement for the child is presented.

TABLE 5.
APPROXIMATE DAILY REQUIREMENTS OF CHILDREN FOR MINERALS*

| Age | Ca | P | Fe | $I_2$ |
|---|---|---|---|---|
| yr. | gm. | gm. | mg. | |
| Infancy | 1.0 | 1.5 | 6 | Trace |
| 1 - 3 | " | " | 8 | " |
| 4 - 6 | " | " | 10 | " |
| 7 - 9 | " | " | 12 | " |
| 10 - 12 | "+ | "+ | 14 | " |
| 13 - 15 | "+ | "+ | 16 | " |
| 15+ | "+ | "+ | 16 | " |

*Tables 5 and 6 adapted from recommendations by the Food and Nutrition Board of the National Research Council. Slight variations made in order to state the amounts necessary for each three-year period after infancy in direct arithmetic progression.

*Magnesium:* Knowledge of specific function is meager but about three-fourths of that in the body is in the skeleton. The body needs are automatically met because of rich vegetable and animal sources.

*Potassium:* Most important electropositive intracellular ion. Deficiency may develop in severe diarrhea. Requirement of 1 to 2 gm. daily is supplied readily in the ordinary diet of children.

*Sodium:* Plays an important role in extracellular fluid regulation. Requirement of 1 to 2 gm. daily for the child is easily supplied in food and as table salt. Deficiency may develop in excess sweating.

Electronegative elements:

*Phosphorus:* Widely distributed in the body as inorganic phosphate, especially in bone and as organic compounds. It is an important intracellular constituent of the body. The actual requirement and retention of the child is somewhat less than for calcium. However, because of its abundance in the diet, greater quantities than are required are usually consumed. The average daily requirement for various ages is presented in table 5.

*Chlorine:* Occurs chiefly as chlorides

and is the most abundant electronegative electrolyte in extracellular fluid and is important in maintenance of acid-base balance and osmotic equilibrium. Under ordinary conditions the child's requirements are met without difficulty. In the pathologic conditions of dehydration, acidosis, and adrenal cortical hormone deficiency—chloride metabolism disturbances occur.

*Sulfur:* Distribution in the body is determined chiefly by the distribution of protein which in turn is found in many different tissues. Diets which supply sufficient amino acid meet the requirement for sulfur in children.

Elements present in important organic complexes such as hemoglobin and thyroxin:

*Iron:* Important in tissue and blood hemoglobin formation also in cytochrome, hence is necessary for growth. The requirement probably varies from 0.4 to 1.0 mg. per kilogram per day in early life to 0.2 to 0.4 mg. for the older child. The recommended daily requirement for children is presented in table 5.

*Iodine:* Essential in the manufacture of thyroxin. It is widely distributed in the earth's surface yet lack of iodine in the soil or water causes goiter in many localities. The difference in iodine intake between individuals living in the goitrous and nongoitrous belts is about 14 micrograms per day. If the diet supplies 40 to 100 micrograms per day (usually expressed as a trace) there is no need to add supplements such as iodized salt (0.01 per cent potassium iodide) to the diet.

Trace elements:

*Fluorine:* Important in bone and tooth structure. Water containing 0.7 parts per million appears to have sufficient fluorine to prevent dental caries and a small enough quantity to prevent mottling of the teeth (fluorosis).

Copper, zinc, manganese, cobalt, silicon, and selenium: Probably of importance in human nutrition but exact roles and requirements not yet determined.

Boron, nickel, aluminum, bromine, and arsenic: Elements present in minute traces in the body but significance in neither human nor animal nutrition has been worked out.

VII. *Vitamins*: These may be termed the glamour factors in nutrition. Nevertheless, in spite of all their popularity they are important and are essential for the maintenance of proper body functioning.

VITAMINS PROVED TO BE ESSENTIAL FOR INFANTS AND CHILDREN

*Sterols with vitamin D activity:* The diets of infants and children are more apt to be deficient in substances having vitamin D activity than any other food essential. There are some 10 sterols which have been found to possess vitamin D activity, the most important of which are activated ergosterol (vitamin $D_2$) from vegetable sources and irradiated 7-dehydrocholesterol (vitamin $D_3$) from animal source. Ultraviolet radiation supplements the vitamin D in the diet. The average daily requirements for children are given in table 6.

TABLE 6.

APPROXIMATE DAILY REQUIREMENTS OF CHILDREN FOR VITAMINS*

| Age | A | Thiamin | Ribo-flavin | Niacin | Ascorbic Acid | D |
|---|---|---|---|---|---|---|
| yr. | I.U. | mg. | mg. | mg. | mg. | I.U. |
| Infancy | 2000 | 0.4 | 0.6 | 4 | 30 | 400 - 800 |
| 1 - 3 | 2500 | 0.6 | 0.9 | 6 | 40 | " |
| 4 - 6 | 3000 | 0.8 | 1.2 | 8 | 50 | " |
| 7 - 9 | 3500 | 1.0 | 1.5 | 10 | 60 | " |
| 10 - 12 | 4000 | 1.2 | 1.8 | 12 | 70 | " |
| 13 - 15 | 4500 | 1.4 | 2.0 | 14 | 80 | " |
| 15+ | 5000 | 1.6 | 2.2 | 16 | 90 | " |

*Tables 5 and 6 adapted from recommendations by the Food and Nutrition Board of the National Research Council. Slight variations made in order to state the amounts necessary for each three-year period after infancy in direct arithmetic progression.

*Ascorbic acid:* This substance is apt to be lacking in the diet of many artificially fed infants. Ascorbic acid is necessary for the structure of intercellular cement ground substance and is concerned with cellular respiration. The recommended daily requirements for various ages are presented in table 6.

*Vitamin A and carotinoid pigments:* Of the various dietary deficiencies which may develop in children those due to the lack of vitamin A or its precursors (most important is beta carotene) may have the most serious results—namely, blindness. The

daily requirement for infants is relatively greater than for adult subjects. The recommended daily allowance for the various ages is given in table 6.

*Vitamin K*: Quinoid structures, vitamin $K_1$ ($C_{31}H_{46}O_2$) from alfalfa and vitamin $K_2$ ($C_{41}H_{56}O_2$) from putrified fish meal appear to be essential for normal prothrombin formation, and these are necessary for proper blood coagulation. The synthesized compound 2-methyl-1, 4-naphthoquinone with the non-proprietary name of menadione appears to have the greatest antihemorrhagic activity. Vitamin K is abundantly supplied in the normal diet but a special need exists for the newborn. This vitamin apparently is synthesized in the normal gastrointestinal tract by bacterial action; however, deficiency may develop when fat absorption is lacking (absence of bile, steatorrhea) and in liver disease.

*Components of the vitamin B complex*—niacin, riboflavin and thiamin: Components of the vitamin B complex are apt to be found in the same food substances, therefore, individuals suffering from deficiency of one component, for example, pellagra, ariboflavinosis, or beriberi are apt to have deficiency of one or both of the others. Each of these factors has been synthesized and each supplies a prosthetic group to form one or more coenzymes which function in an enzyme system concerned with cellular respiration. The suggested daily requirements for children are given in table 6.

Vitamins which may be of importance in the nutrition of infants and children are:

*Other components of the vitamin B complex*—pyridoxine, pantothenic acid, biotin, choline, inositol, and para-aminobenzoic acid. These substances are known to be of significance nutritionally from work with experimental animals. Although there is some evidence to indicate that they may be of importance in human nutrition, the specific needs for infants and children are not known, hence, the daily requirements are not known.

*Vitamin E:* Alpha tocopherol is the most important of some forty substances which possess vitamin E activity. In spite of many studies the actual human need is no more than conjectured.

Vitamins not clearly defined even from studies with experimental animals are:

*Factors of the vitamin B complex*—vitamin $B_3$ (pigeons), vitamin $B_4$ (rats and chicks), vitamin $B_5$ (pigeons), factor U, R, and S (chicks), cartilage growth factor (chicks), folic acid or nucleotide (chicks, Lactobacillus casei) and kidney residue (chicks).

*Miscellaneous vitamins:* Citrin or vitamin P containing two components, hesperdin and eriodictin is said to be concerned with capillary resistance. Grass juice factor is concerned with growth in guinea pigs; "milk factor" with stomach hemorrhages and ulcers in guinea pigs; vitamin L, a factor for lactation in rats; and vitamin M, prevents anemia, leukopenia, and weight loss in monkeys.

VIII. *Miscellaneous factors in nutrition:* For complete fulfillment of the nutritional needs of children the following dietary factors must be considered: (1) Roughage; (2) digestibility; (3) satiety; (4) proper food preparation for cleanliness, appetite appeal, and palatibility; (5) availability — economic and geographic; (6) pigments, antioxidents and enzymes—human dietary significance not known; (7) possible synthesis of vitamins in gastrointestinal tract; (8) psychologic factors—one of the most common problems in modern practice of medicine in children mostly because of parental over-anxiety.

## SUMMARY AND CONCLUSIONS

The physician may become frustrated at times by the maze resulting from the mass of new discoveries, temporary overemphasis on individual dietary factors, vague terminology such as "submarginal," "subclinical," "combined deficiency," "twilight zone," and "hidden hunger" states, and from the sweeping "educational" program attempted by some of the commercial advertisers. From a practical viewpoint, the daily requirement of the growing child and the daily diet which will satisfy these requirements may be stated very simply. The

following recommendations are adapted from those of the Food and Nutrition Board of the National Research Council:

| | |
|---|---|
| Protein | 2 gm. per kg. body weight |
| Fat | |
| Carbohydrate | Sufficient to meet caloric needs |
| Minerals | |
| Calcium | 1.0 gm. |
| Phosphorus | 1.5 gm. |
| Iron | 16.0 mg. |
| Iodine | Trace |
| Vitamins | |
| A | 5000 I.U. |
| Thiamin | 1 mg. |
| Riboflavin | 2 mg. |
| Nicotinic acid | 10 mg. |
| Ascorbic acid | 60 mg. |
| D | 400-800 I.U. |

Composition of a diet which satisfies the basic nutritional needs of the growing child:

| | |
|---|---|
| Milk | 3/4 to 1 quart daily |
| Meat (fresh, lean), or fish | 1 serving daily or 5-6 per wk. |
| Eggs | 1 daily or 5-6 per wk. |
| Vegetables (1 raw, 1 pigmented) | 2 or more servings daily |
| Fruits (1 fresh) | 2 or more servings daily |
| Butter | 2 tsp. or more daily |
| Liver | 1 serving or more per wk. |
| Bread and cereals (whole grain) | Sufficient to meet caloric need |
| Cod Liver oils | 1 tsp. or equivalent daily |
| Salt (iodized) | for seasoning |

In conclusion, one of the valuable aids to the physician in the appraisal of a child's state of nutrition is the response noted to the correction of a defective dietary regimen or to the administration of specific food factors, such as amino acids, fat, certain minerals or vitamins. In the interpretation of these so-called therapeutic tests in the diagnosis of nutritional deficiency in some instances clinical changes may be detected within a few days, e. g., in the deficiencies due to vitamin K, ascorbic acid, vitamin D, nicotinic acid, riboflavin, thiamin, vitamin A (nyctalopia). In other instances changes may require weeks to months to me discernible, for example, in the nutritional disorders resulting from the lack of vitamin D, protein, iodine, iron, vitamin A, and in still others many months may pass before results or restitution of the condition to normal can be expected, for example, in the xerosis and skin eruptions due to lack of vitamin A, and in the dermatoses which may be associated with disturbances in unsaturated fatty acid metabolism.

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## RHEUMATIC HEART DISEASE*

### A STUDY OF 167 CASES AMONG 12,317 AUTOPSIES PERFORMED AT THE CHARITY HOSPITAL IN NEW ORLEANS DURING TEN YEARS

IWAO UYEDA, M. D.

NEW ORLEANS

Because of apparent or real differences in symptomatology, course, and prognosis of rheumatic heart disease as it occurs in different parts of the world,[1-11] it is important to review features which characterize this disease in various geographical areas. The present study is concerned with a partial analysis of cases with rheumatic heart disease encountered among routine autopsies performed at Charity Hospital in New Orleans during the past ten years.

METHOD OF INVESTIGATION

Twelve thousand three hundred and seventeen autopsy records in the period from January 1, 1935 to January 1, 1945, were reviewed in search of proved cases of rheumatic heart disease. Such a statistical survey should yield more accurate knowledge of incidence than clinical diagnoses, since the latter are not always subject to accurate demonstration or objective evaluation; it has been suggested[12] that the supposed rarity of rheumatic infection in the southern United States may be due in part to failure of recognition. Validity of autopsy statistics is enhanced by the fact that in a chronic disease such as rheumatic fe-

*Condensation of thesis presented to Department of Pediatrics, Tulane University School of Medicine, May 1945.

ver, the majority of patients afflicted probably die in hospitals. Hedley[13] noted that 94.3 per cent of 1,020 deaths from rheumatic heart disease in Philadelphia occurred in hospitals.

For the purpose of this study, only cases with a definite pathologic diagnosis of rheumatic heart disease were accepted. None were included in which clinical diagnoses of rheumatic heart disease could not be confirmed pathologically, in which only "suspicious" or "atypical" lesions were noted, or in which the pathologist was unwilling to make a definite commitment as to presence of typical rheumatic lesions.

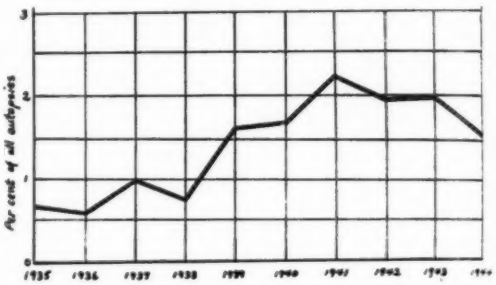
Although mitral stenosis has been considered to be almost always rheumatic in origin,[14, 15] its presence at autopsy was not considered adequate for inclusion in this study unless it was accompanied by a rheumatic history and the pathologist's statement of a typical rheumatic character. Data for 1944 are yet incomplete and may be subject to criticism or revision. Figures in this study are considered conservative, since only anatomically proved cases were included.

INCIDENCE

Chart I represents the number of cases by years recorded in routine autopsies. During this ten year period, rheumatic heart disease was diagnosed anatomically in 1.35 per cent of all autopsies. Of particular interest is the increase of rheumatic lesions at autopsy since 1939 and the persistently higher incidence since then. It is noteworthy that these figures are considerably greater than those often quoted for the South in general. This may be either due to actual increasing incidence, as hinted by Bailey,[1] or may merely be in confirmation of the aphorism "Was man weiss, sieht man." Discussion of this point is not the purpose of this paper, however. In Charity Hospital at New Orleans, as in many other Southern hospitals, it seems obvious that rheumatic fever is being diagnosed clinically more frequently now than it was eight or ten years ago. Houston[8] reported 88 rheumatic cases among 115,213 admissions to the Charity Hospital at New Orleans for the period 1922-1927 (0.076 per cent), whereas Bruno and Engelhardt[2] reported 875 rheumatic cases among 187,987 admissions for the period 1939-1941 (0.46 per cent); the latter incidence is six times greater than that of the earlier report.

CHART I

Percentage distribution by years of 167 cases of rheumatic heart disease among routine autopsies at the Charity Hospital in New Orleans from January 1, 1935 to January 1, 1945.




Incidence of Rheumatic Heart Disease Among Routine Autopsies

| Year | Number Autopsies | Rheumatic Cases | Per cent Rheumatic |
|---|---|---|---|
| 1944 | 890 | 14 | 1.57 |
| 1943 | 1001 | 20 | 1.98 |
| 1942 | 872 | 17 | 1.94 |
| 1941 | 1241 | 29 | 2.23 |
| 1940 | 1367 | 23 | 1.68 |
| 1939 | 1486 | 24 | 1.61 |
| 1938 | 1320 | 10 | .75 |
| 1937 | 1246 | 12 | .96 |
| 1936 | 1547 | 9 | .58 |
| 1935 | 1347 | 9 | .66 |
| Total | 12,317 | 167 | |

The question as to whether these rheumatic subjects were natives of the South or whether they had migrated from the North in an effort to find environment supposedly more congenial to their disease cannot be answered. Among the 167 cases analyzed, the place of birth was stated in 27; no single instance was noted in which the patient had migrated from the Northern states. All were natives of Louisiana with

the exception of one, who was from Florida. Data regarding racial background are incomplete.

Several criteria have been used by investigators for expressing incidence. Most frequently quoted are morbidity figures based on clinical diagnoses alone; when observations at autopsy have been tabulated, many lesions only suggestively rheumatic have often been included.

The unusually high incidence of 9.1 per cent among 5,215 autopsies reviewed at the Boston City Hospital by Davis and Weiss[16] resulted from consideration of clinical and morphologic data together. This was appreciably higher than any previous or later figures encountered. Figures from our present study are in fairly close agreement with those reported by Longcope.[17] He found rheumatic cardiac lesions in 1.66 per cent of 8,164 autopsies at Johns Hopkins Hospital in the period from 1908 to 1929 and concluded that the disease was as common in hospitals in Baltimore as in those of Boston.

In the past five years, rheumatic heart disease has been diagnosed anatomically 103 times among 5,371 routine autopsies at Charity Hospital in New Orleans, an incidence of 1.91 per cent. In view of the rigid criteria used to obtain this figure, the New Orleans area does not appear to have as low an incidence rate as is commonly supposed.

### RACE, SEX AND AGE

Many figures[2, 17-20] have been quoted on incidence of rheumatic fever by race and sex. In this series there were 83 cases among whites and 84 among colored patients (table 1); 56.3 per cent were male and 43.7 per cent female. These figures are in contrast to the general concept that rheumatic fever is more common in females. Another notable observation was the earlier age at death among females, being 29.2 years as opposed to average age of 36.4 years in males (table 2, chart 2). Fifty-seven and a half per cent of female rheumatics died by 30 years of age, whereas for males the cumulative death by 30 years of age was 40.4 per cent.

TABLE 1

INCIDENCE BY RACE AND SEX OF 167 CASES OF RHEUMATIC HEART DISEASE AMONG ROUTINE AUTOPSIES AT THE CHARITY HOSPITAL IN NEW ORLEANS FROM JANUARY 1, 1935 TO JANUARY 1, 1945.

| White | | Colored | |
|---|---|---|---|
| Female | 32 | Female | 41 |
| Male | 51 | Male | 43 |
| | 83 | | 84 |

TABLE 2

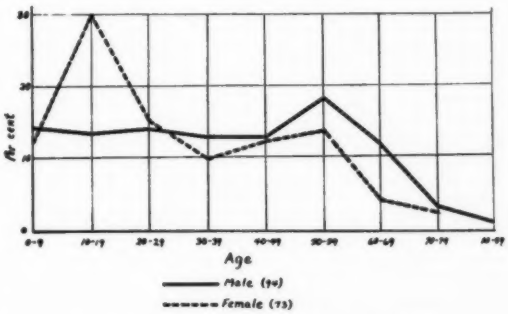
MEAN AGE AT DEATH BY RACE AND SEX OF 167 CASES OF RHEUMATIC HEART DISEASE AMONG ROUTINE AUTOPSIES AT THE CHARITY HOSPITAL IN NEW ORLEANS FROM JANUARY 1, 1935 TO JANUARY 1, 1945.

| White | | Colored | |
|---|---|---|---|
| Male | 40.43 yrs. | Male | 31.72 yrs. |
| Female | 33.37 yrs. | Female | 26.06 yrs. |

| | |
|---|---|
| White | 37.69 yrs. |
| Colored | 28.96 yrs. |
| White & Colored | 33.30 yrs. |
| Male (W & C) | 36.45 yrs. |
| Female (W & C) | 29.26 yrs. |

CHART II

Comparative percentage distribution by age decades of 94 cases of male with 73 cases of female patients having rheumatic heart disease among autopsies at the Charity Hospital in New Orleans from January 1, 1935 to January 1, 1945.




Comparative Deaths by Age Decades in Male and Female

| | Male | | Female | |
|---|---|---|---|---|
| Age | Deaths | Per cent | Deaths | Per cent |
| 0-9 | 13 | 13.83 | 9 | 12.32 |
| 10-19 | 12 | 12.77 | 22 | 30.13 |
| 20-29 | 13 | 13.83 | 11 | 15.06 |
| 30-39 | 12 | 12.77 | 7 | 9.58 |
| 40-49 | 12 | 12.77 | 9 | 12.32 |
| 50-59 | 17 | 18.08 | 10 | 13.68 |
| 60-69 | 11 | 11.70 | 3 | 4.18 |
| 70-79 | 3 | 3.19 | 2 | 2.73 |
| 80-89 | 1 | 1.06 | 0 | |
| | 94 | | 73 | |

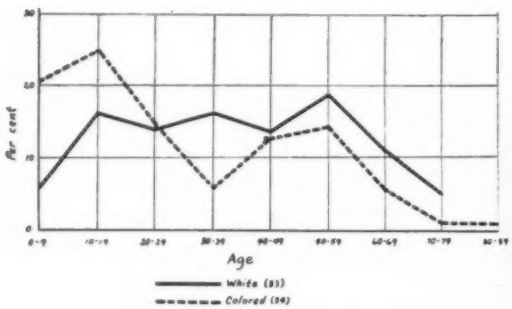
The age at death confirmed the general opinion that rheumatic fever runs a shorter and more severe course in the colored race (chart 3), the mean age at death for the colored being 28.9 years in contrast to 37.6 years for the whites (table 2). Fifty-nine and a half per cent of deaths among colored rheumatics occurred in the first three decades, and 36 per cent of deaths were noted among the whites. A secondary rise in mortality figures among negroes was seen in the age group from 50-59 years, whereas among whites the age at death was fairly distributed for the first half century, with the maximum number of fatalities in the period from 50-59 years (chart 3). The peak age at death for negroes was in the second decade and among whites in the sixth decade, with the second decade a close second. Hedley[21] obtained the highest mortality incidence among negroes in the second decade, and found a gradual rise among whites to a peak in the age group from 40-49 years. These figures further support the belief that this disease is more malignant in the colored race.

Considering all cases, the mean age at death was 33.3 years (table 2), a slightly higher figure than that of 28.9 years reported by Hedley[13] in Philadelphia and of 28.2 years by Coombs[22] in Great Britain. DeGraff and Lingg,[23] however, reported that the mean age at death of 644 patients at Bellevue Hospital was 33 years; Willius[24] gave 32 years as the mean age of 160 patients who died of rheumatic heart disease at The Mayo Clinic.

Cardiac deaths under 40 years of age have almost invariably been considered

CHART III

Comparative percentage distribution by age decades of 83 cases of white and 84 cases of colored patients having rheumatic heart disease among routine autopsies at the Charity Hospital in New Orleans from January 1, 1935 to January 1, 1945.

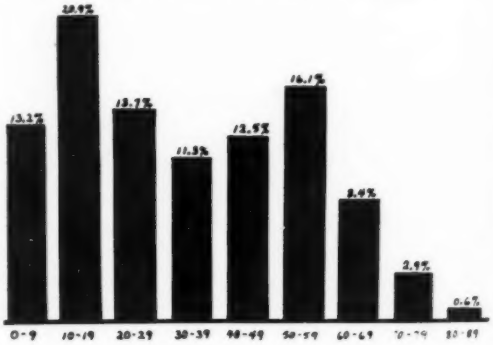



Comparative Deaths by Age Decades in White and Colored

| Age | White | | Colored | |
|---|---|---|---|---|
| | Deaths | Per cent | Deaths | Per cent |
| 0-9 | 5 | 6.03 | 17 | 20.24 |
| 10-19 | 14 | 16.87 | 21 | 25.00 |
| 20-29 | 11 | 13.25 | 12 | 14.29 |
| 30-39 | 14 | 16.87 | 5 | 5.95 |
| 40-49 | 11 | 13.25 | 10 | 11.91 |
| 50-59 | 15 | 18.07 | 12 | 14.28 |
| 60-69 | 9 | 10.84 | 5 | 5.95 |
| 70-79 | 4 | 4.82 | 1 | 1.19 |
| 80-89 | 0 | | 1 | 1.19 |
| | 83 | | 84 | |

CHART IV

Percentage distribution by age decades of 167 cases of rheumatic heart disease among routine autopsies at the Charity Hospital in New Orleans from January 1, 1935 to January 1, 1945.




Deaths by Age Decades

| Age | Deaths | Per cent |
|---|---|---|
| 0-9 | 22 | 13.19 |
| 10-19 | 35 | 20.96 |
| 20-29 | 23 | 13.78 |
| 30-39 | 19 | 11.38 |
| 40-49 | 21 | 12.58 |
| 50-59 | 27 | 16.15 |
| 60-69 | 14 | 8.39 |
| 70-79 | 5 | 2.98 |
| 80-89 | 1 | .59 |
| | 167 | |

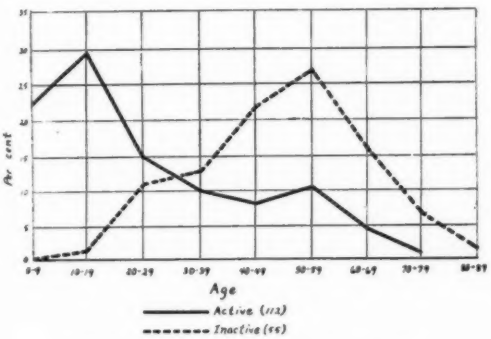
rheumatic.[9] The greatest number of deaths (20.9 per cent of the total deaths) occurred during the second decade (chart 4). Over 10 per cent of the deaths had occurred by ten years, over 30 per cent by 20 years, nearly 50 per cent by 30 years, and almost 60 per cent by the fortieth year of age.

PATHOLOGIC FINDINGS

These 167 cases of rheumatic heart disease were considered as "active" or "inactive," according to the type of lesion observed at autopsy. For the active group, criteria were the presence of Aschoff nodules, small bead-like vegetations on valve leaflets, acute fibrinous pericarditis, or a swollen and edematous mitral valve associated with one of the other active rheumatic lesions mentioned. Classification as "inactive" was based upon mitral stenosis, thick mitral valve and shortened chordae tendenae, chronic adhesive pericarditis, or myocardial fibrosis associated with one of the other inactive rheumatic lesions. Weight of the heart was not considered in differentiating from inactive cases.

CHART V

Comparative percentage distribution by age decades at time of death of 112 cases of active and 55 cases of inactive rheumatic heart disease among routine autopsies at the Charity Hospital in New Orleans from January 1, 1935 to January 1, 1945.




Comparative Deaths by Age Decades in Active and Inactive Cases

| | Active | | Inactive | |
|---|---|---|---|---|
| Age | Deaths | Per cent | Deaths | Per cent |
| 0-9 | 23 | 20.53 | 0 | 0 |
| 10-19 | 33 | 29.47 | 1 | 1.82 |
| 20-29 | 17 | 15.19 | 6 | 10.91 |
| 30-39 | 12 | 10.71 | 7 | 12.73 |
| 40-49 | 9 | 8.03 | 12 | 21.82 |
| 50-59 | 12 | 10.71 | 15 | 27.27 |
| 60-69 | 5 | 4.47 | 9 | 16.36 |
| 70-79 | 1 | .89 | 4 | 7.27 |
| 80-89 | 0 | | 1 | 1.82 |
| | 112 | | 55 | |

Signs of rheumatic activity have been noted in a large proportion of hearts of individuals as old as 40 years who died of rheumatic heart disease,[25] and it has also been stated[26] that signs of active myocarditis are present in fully 80 per cent of fatal cases. Sixty-seven per cent of these 167 cases showed active manifestations while the remaining 33 per cent showed only inactive lesions (chart 5). In 107 cases, death was attributable directly to rheumatic heart disease (table 3).

Age at death of active and inactive cases varied greatly, the mean age for the active group being 23.6 years and for the inactive 52.2 years. Peak age at death among active cases was in the second decade, and of the inactive cases in the sixth decade (chart 5). By age 30, 65 per cent of deaths had occurred in the active group, whereas there were only 12.7 per cent in the inactive group. The age at death in relation to first manifestation of rheumatic fever or rheumatic heart disease cannot be stated from the data available.

Rheumatic lesions were noted as merely incidental in 39, or 24 per cent of the cases (table 3). No clinical diagnosis of rheumatic fever, rheumatic heart disease or subacute bacterial endocarditis was made for any of these cases; and unrelated disease adequate to explain death was discovered in each one.

TABLE 3

CAUSES OF DEATHS OF 167 CASES OF RHEUMATIC HEART DISEASE AMONG ROUTINE AUTOPSIES AT THE CHARITY HOSPITAL IN NEW ORLEANS FROM JANUARY 1, 1935 TO JANUARY 1, 1945

| | Cases | Per cent |
|---|---|---|
| Rheumatic heart disease | 107 | 63.17 |
| Bacterial endocarditis superimposed upon rheumatic heart disease | 21 | 12.65 |
| Other causes with rheumatic heart disease as an incidental finding | 39 | 24.18 |

There were 21 cases in which bacterial

endocarditis was superimposed on rheumatic lesions; seven were in white males, eight in white females, three in colored males and three in colored females. Among these 21 were 15 examples of subacute bacterial endocarditis and six of suppurative endocarditis, three of the latter in white females and one each in white male, colored male and colored female. The mean age at death of these patients was considerably higher than that published by Hedley,[13] who reported 29.2 years as the mean age among 81 fatal cases of subacute bacterial endocarditis superimposed on rheumatic heart disease in the Philadelphia hospitals. In the present study, the mean age at death was 33.4 years. Laws and Levine[27] found a mean of 38.2 years, however, in a group of 43 such cases.

In this entire series of 167 cases, involvement of the mitral valve was not mentioned in nine; five of these had aortic stenosis alone and in the other four no valvular involvement was described. Severe, extreme stenosis of the mitral valve was seen in 30 patients (17.9 per cent); 53.3 per cent were in males and 46.7 per cent in females; 80 per cent were white, 20 per cent colored. In 42 (25 per cent) there were both mitral and aortic valves involved; in eight (4.8 per cent) the mitral, aortic and the tricuspid valves were involved; in seven (4.2 per cent) the mitral and the tricuspid valves were affected; and there was one instance of pulmonary valve involvement (0.6 per cent). Pericardial disease was detected in 45 cases (26.9 per cent). Of some interest is the associated luetic aortitis seen in eight cases (4.8 per cent) and diffuse glomerulonephritis in five cases (2.9 per cent); the mean age at death for the latter was 26 years of age.

TABLE 4

PATHOLOGIC FINDINGS IN 167 CASES OF RHEUMATIC HEART DISEASE AMONG ROUTINE AUTOPSIES AT THE CHARITY HOSPITAL IN NEW ORLEANS FROM JANUARY 1, 1935 TO JANUARY 1, 1945

Valvular Involvements

| | Cases | Per cent |
|---|---|---|
| Mitral | 158 | 94.01 |
| Mitral and aortic | 42 | 25.15 |
| Mitral, aortic and tricuspid | 8 | 4.79 |
| Mitral and tricuspid | 7 | 4.19 |
| Mitral, aortic and pulmonic | 1 | .59 |
| Aortic alone | 5 | 2.98 |
| Valvular involvement not mentioned | 4 | 2.34 |
| Mitral stenosis | 30 | 17.96 |
| Male | 16 | 53.33 |
| Female | 14 | 46.67 |
| White | 24 | 80.00 |
| Colored | 6 | 20.00 |
| Aortic stenosis | 12 | 7.18 |
| Male | 12 | 100.00 |
| Female | 0 | 0.0 |
| White | 9 | 75.00 |
| Colored | 3 | 25.00 |

Pericarditis

45 ..... 26.94

Associated Luetic Aortitis

8 ..... 4.79

Associated Glomerulonephritis

5 ..... 2.98

## CONCLUSION

There are great variations in the methods used for determining the incidence of rheumatic heart disease. This is accounted for in part by the fact that rheumatic fever is not a reportable disease, and thus incidence figures must be compiled from hospital records. There are no uniform criteria by which hospital incidences are determined. Figures obtained from different reports but from the same locality may vary considerably. This series from the Charity Hospital in New Orleans included only those cases which were definitely classified as "rheumatic" by the pathologist. Obviously many examples of "clinical" rheumatic heart disease must have been omitted from this study. Nevertheless, the incidence of rheumatic heart disease in this area, evaluated on the basis of distinctive pathology, is higher than is commonly supposed, and not strikingly lower than in the North. The danger, however, of drawing too definite a conclusion on the basis of this comparatively small series is still present.

The number of cases of fatal rheumatic heart disease can give only a hint of the incidence in any community. If rheumatic fever in the South is milder than in the North[1, 2, 8, 17] and therefore less apt to terminate fatally, then the same autopsy incidence figure would indicate a greater incidence of rheumatic heart disease in the living population of the South than it would in the North. Perhaps too much importance has been placed on the geographic location as a conditioning factor in the inci-

dence of rheumatic fever and not enough emphasis on the strictly local characteristics, such as the urban or rural communities, relative age of the population, economic status of the people, and local changes in humidity and temperature.

SUMMARY

Twelve thousand three hundred and seventeen routine autopsies from the Charity Hospital of Louisiana at New Orleans were reviewed for anatomically proved cases of rheumatic heart disease. For the ten year period from January 1, 1935 to January 1, 1945, 167 cases were discovered, 1.35 per cent. Of these, 103 occurred in the period from January 1, 1940 to January 1, 1945, an incidence of 1.91 per cent rheumatic heart disease among routine autopsies in the past five years. These figures approach very closely those obtained from similar studies carried out in Northern cities.

There were no extreme, wide racial differences in incidence, but age at death and cumulative deaths by age decades indicate that rheumatic fever runs a shorter and a more severe course among negroes. It may also be inferred that rheumatic fever is more malignant in females.

The mean age at death did not vary significantly from figures obtained from the Northern cities, indicating that rheumatic fever may not necessarily be more benign in the South.

Of the 167 rheumatic heart disease cases diagnosed anatomically, 67 per cent showed evidence of active rheumatic infection. Comparisons of active and inactive groups show that the critical period in the rheumatic patients are the early years after the onset of the disease, when exacerbations are most likely to occur. Rheumatic heart disease was found to be the cause of death in 63 per cent of the cases; in 24 per cent rheumatic lesions were incidental observations at autopsy; and in 12.6 per cent bacterial endocarditis superimposed upon rheumatic lesions was the cause of death.

Twenty-one cases were found in which bacterial endocarditis was superimposed upon rheumatic lesions. In 94 per cent of the cases the mitral valve was involved, both the mitral and the aortic valves in 25 per cent, the involvement of the mitral, aortic and the tricuspid valve in 4.8 per cent, and the mitral and the tricuspid valves in 4.2 per cent of the cases. One instance of pulmonary valve involvement was seen (0.6 per cent). Extreme mitral stenosis was noted in 17.9 per cent of the cases and pericardial involvement in 26.9 per cent. Associated luetic aortitis was present in 4.8 per cent and glomerulonephritis in 2.9 per cent of the cases.

From this study, it appears that geographic location alone is not a very important factor in estimating incidence of rheumatic fever. The New Orleans area apparently presents a much higher incidence of rheumatic heart disease than is commonly credited to that region.

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# HEMOLYTIC ANEMIA OF THE NEWBORN (ERYTHROBLASTOSIS FETALIS)*

## REPORT OF CASES

RUTH G. ALEMAN, M. D.†

NEW ORLEANS

Most of the available knowledge on the subject of hemolytic anemia of the newborn in its present concept dates back only to 1940. At that time Landsteiner and Wiener[1] reported the existence of a newly recognized antigenic constituent of the red blood cells of the majority of human beings which they called Rh because of its initial recognition in the red blood cells of the rhesus monkey.

While antibodies against this antigen do not occur normally in human plasma, it has been shown that under certain conditions of stimulation they may exist in persons devoid of antigen.

Based on the recent work of Levine it is now established that in hemolytic anemia of the newborn the fetus inherits the Rh factor from a father with Rh positive blood. "In more than ninety per cent of the cases the factor is present in the father's blood, Rh+, absent in the mother's blood, Rh—, while the blood of the fetus must possess it, Rh+."[2]

The essential steps in the development of hemolytic anemia of the newborn are: "(1) The response of the Rh— mother to the foreign Rh+ fetal blood by the production of anti-Rh agglutinins and (2) the continuous passage of maternal anti-Rh agglutinins through the placenta to react with and hemolyze the susceptible fetal Rh+ blood."[2]

The Rh agglutinogen (fetal blood) passes through some break in the placental barrier to the maternal circulation and stimulates the production of antibodies which diffuse back into the fetus and destroy its red blood cells,—only if the maternal and fetal blood are incompatible.

In some cases postpartum anemias, out of proportion to the blood lost at the time of delivery, occur. The possibility of the isoimmunization mechanism working in reverse must be thought of. With an Rh positive mother carrying an Rh negative fetus, through intermixing of maternal and fetal blood, the fetus may produce Rh antibodies capable of attacking the mother's red blood cells and causing anemia.[3]

### CASE No. 1

Baby R., the fourth sibling, was delivered normally at term, weighing 8 pounds, 7 ounces. He was referred immediately after birth because of intense jaundice, with numerous purpuric spots scattered over the entire body. The right scrotum was filled with blood. The corneal conjunctivae bulged with hemorrhage. There was both splenomegaly and hepatomegaly. The respiration was rapid and irregular. The urine was markedly bile stained. See plate 1 for laboratory findings, treatment and progress.

It is interesting to note that the first two children in this family were entirely normal while the third child, soon after birth, developed a profound anemia which necessitated immediate blood transfusion and subsequent iron and liver therapy.

The blood of this entire family was submitted to Drs. Philip Levine and John Davenport for studies of the Rh factor. Both agree that "This

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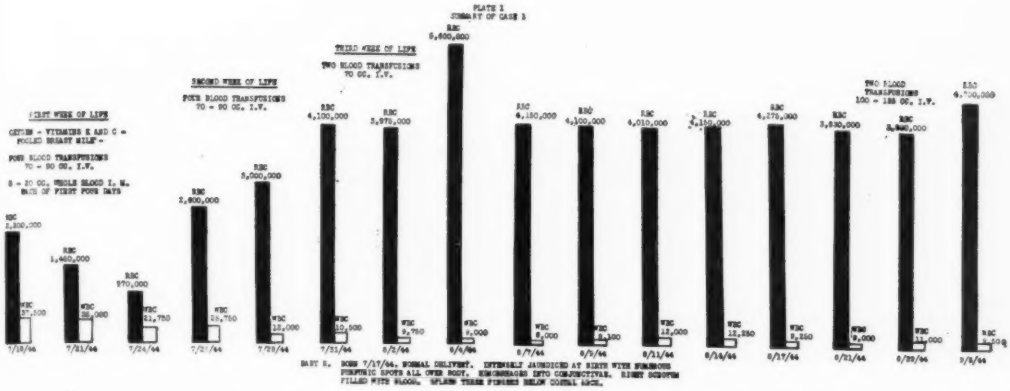
*Presented at Hotel Dieu Staff meeting, New Orleans, La., June 25, 1945.

†From the Department of Pediatrics, Louisiana State University School of Medicine.




case presents a typical history and serological findings of Rh iso-immunization resulting from pregnancy. The mother is Rh— while the father and the four boys are all Rh+. The father is $Rh_1$, $Rh_2$. This, together with the fact that all four children are also Rh+, is strong evidence that the father is homozygous (Rh Rh)."

CASE No. 2

Baby P., full term, first sibling, weighing 6 pounds, 2 ounces, was delivered by low forceps. Intense jaundice was noted the first day of life. This newborn was referred on the second day because of jaundice. The red cell count at this time was 915,000, white blood count 30,000. The mother was Rh negative, the baby was Rh positive. The father, being overseas in service, was not tested. For details see plate 2.

CASE No. 3

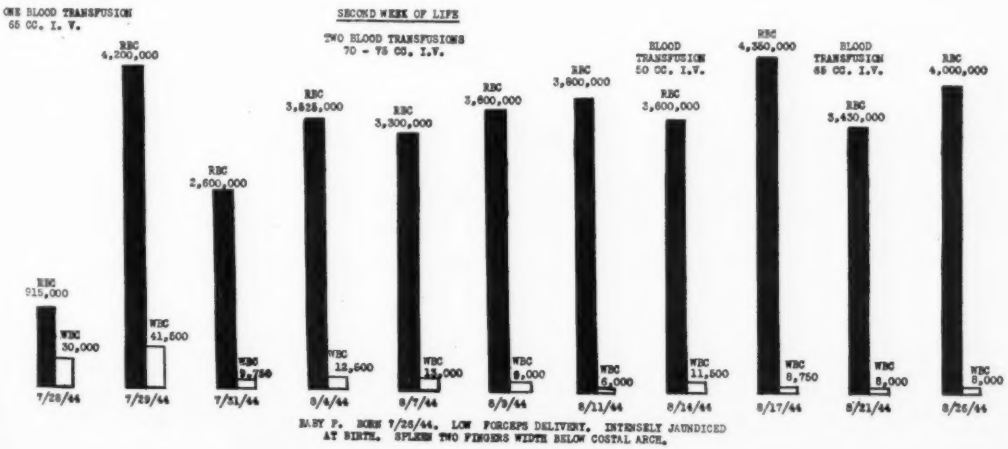
Baby W., first sibling, full term, normal delivery, weighing 8 pounds, 7 1/4 ounces, was markedly jaundiced on second day of life. When Rh studies were made, the mother was found negative, the father and baby Rh positive. See plate 3.

SUMMARY

Three cases of hemolytic anemia of the newborn treated in 1944 are reported. Two cases presented the extreme picture of this condition, jaundice, marked anemia, edema, erythroblastemia. The third case was mild in type. In two cases (case 1 and case 3) the Rh factor was typical (father and baby Rh positive, mother Rh negative). In case 2 the mother was Rh negative, the baby Rh positive. The father who was in service was not examined.

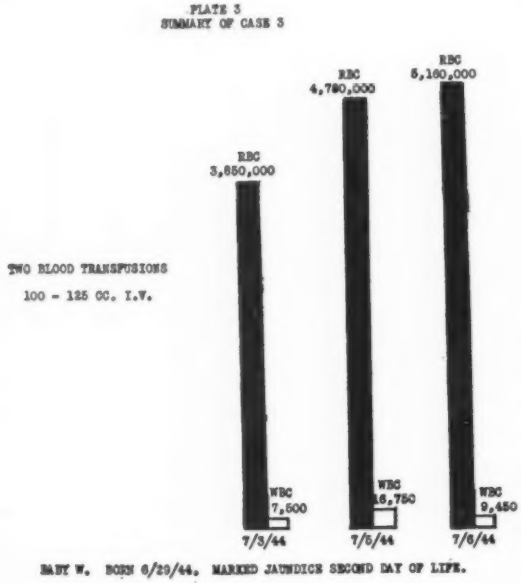
The treatment consisted of transfusions with Rh negative blood, vitamin K, vitamin C and oxygen. All three babies recovered, were normal at two months of age and their

PLATE 2
SUMMARY OF CASE 2




BABY P. BORN 7/28/44. LOW FORCEPS DELIVERY. INTENSELY JAUNDICED AT BIRTH. SPLEEN TWO FINGERS WIDTH BELOW COSTAL ARCH.

subsequent course for the first year of life excellent.




CONCLUSION

The subject of hemolytic anemia of the newborn should be of interest not only to pediatricians but to all medical practitioners, especially obstetricians, for early recognition and treatment are important. My purpose in presenting these cases is to emphasize the fact that hemolytic anemia of the newborn is not necessarily fatal. On the contrary, with early treatment the outlook is often optimistic.

Medical science may count another triumph in the recognition and cure of hemolytic anemia of the newborn. The studies of the Rh factor and the method of cure are too recent to predict the behavior of the blood in later life in relation to so-called "bleeders"—but the important thing to remember is that babies born with hemolytic anemia need not die. Early and proper treatment will often save them.

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*For an exhaustive study with report of fifty-five cases see also Leonard, Martha F.: Hemolytic disease of the newborn, J. Pediat., 27:249, 1945.

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# FATAL MEDITERRANEAN (COOLEY'S) ANEMIA

G. RICHARDA WILLIAMSON, M. B. Ch. B. (Edin.)†
and
RENA CRAWFORD, M. D.††
NEW ORLEANS

The occurrence of fatal Mediterranean erythroblastic anemia in the fourth, the seventh and the eleventh child born of Sicilian parents is reported in this paper. Observations on one of these patients, the eleventh sibling, are presented here in detail, whereas comments on the remaining two victims of the disease are outlined rather briefly.

Mediterranean or Cooley's erythroblastic anemia was identified by Cooley, Lee and Witwer approximately 25 years ago as a familial disease entity peculiar to the children of inhabitants of the north shore of the Mediterranean Sea, especially those of Italian and Greek parentage. The first clue which led to the identification of Mediterranean anemia was the distinct resemblance to the Mongoloid race which these patients may acquire during the course of the disease. This characteristic is due partly to the development of a yellowish pigmentation of the skin and partly to progressive thickening of the malar eminences, and of the skull.

Since the original report which identified Mediterranean anemia appeared many articles dealing with the disease have been published. References to some of these publications are recorded in the list of literature included in this paper. As a result of these and other publications detailed information relative to the characteristics of the disease is available.

†From the Department of Pediatrics, Tulane University, and ††Department of Pediatrics, Louisiana State University, New Orleans, Louisiana.

In the more severe examples of Mediterranean anemia the initial symptoms include pallor, anorexia, weakness and increasing prominence of the abdomen due chiefly to progressive enlargement of the spleen. Later, hypertrophy of the liver augments the abdominal protrusion. The disease tends in general to pursue a very uniform course which usually features no special complications. Long stationary periods during which the patients are comparatively comfortable may be interrupted, however, by exacerbations associated often with infection, but definite hemolytic crises and joint pains are not a part of the clinical picture.

In the more advanced stages of the disease the erythrocytes are greatly reduced in number and the color index is low. An abundance of nucleated red cells in every stage of development and the presence of many hypochromatic macrocytes measuring 15 microns or more in diameter constitute two additional characteristics of the disease. Microcytes, poikilocytes, reticulocytes and fragments of red cells are also abundant in the stained smear. The fragility of the erythrocytes in hypotonic saline solutions spans a wider range of resistance than normal. Some investigators consider this an important and constant characteristic of the disease.

The leukocyte count is constantly elevated but otherwise it usually discloses nothing distinctive. In advanced stages of the disease the increase in the number of granulocytes and of immature cells may be sufficient to suggest myeloid leukemia. The platelet count is not greatly altered except that a sustained increase in their number may follow splenectomy.

During the course of the disease bone changes become increasingly conspicuous on roentgenograms. The alterations become evident first in the small bones of the hands and feet but later other parts of the skeleton are noticeably involved. The cortex of the long bones becomes thin owing presumably to the constant pressure of the proliferating marrow. This change widens the bones and gives them a strikingly transparent appearance on the x-ray plate. In addition the trabeculae become relatively conspicuous especially in the region of the ends of the diaphyses. Later the flat bones, and particularly the skull are involved. The calvarium undergoes great thickening and becomes increasingly porous owing to expansion of its medullary portion. Accompanying this change the inner and outer tables of the cranium, particularly the latter, become very thin and the trabeculae spaning the intervening space are eventually converted into delicate spicules which on the x-ray film resemble radiating bristly hairs attached to the inner plate of the calvarium.

The preceding discussion applies to the more serious and readily identified forms of Mediterranean anemia. Recently evidence has been presented indicating that the disease spans a continuous range of severity varying from extremely mild to very grave forms. Apparently the former outnumber the latter. This view is based on the fact that studies of the blood of siblings and parents of patients with well advanced Mediterranean anemia have disclosed a relatively high familial incidence of individuals who present some evidence of milder forms of the disease. The blood changes found in these patients include varying degrees of refractory hypochromic anemia associated with the presence of microcytes, hypochromatic macrocytes, reticulocytes, stippled cells, and increased resistance to hypotonic sodium chloride solutions. Demonstration of the presence of a few or all of these characteristics in the blood of relatives of the patients with severe Mediterranean anemia aids in the recognition of the milder forms of the disease.

The severe variety of Mediterranean anemia with conspicuous changes involving the blood and bones progresses to a fatal issue in the course of a few years, whereas the very mild forms of the disease may have little or no apparent effect on the health and longevity of the patient.

Since the disease does not respond to any known form of treatment the therapy is limited chiefly to transfusions which merely

provide temporary benefits. Splenectomy is commonly followed by an increase in the number of nucleated red blood cells and the procedure does not stop the progress of the anemia. Consequently this therapeutic measure probably should not be recommended. Furthermore the application of deep x-ray therapy to the spleen and long bones is of doubtful value.

The patient with whom this communication is chiefly concerned was the eleventh child born of Sicilian parents. At the time of birth (July, 1931), she weighed eight pounds. She started to walk and to talk shortly before her first birthday and she was breast fed for two years. Her health appeared to be excellent until the age of nine months, when a definite pallor associated with the development of a yellow discoloration of the skin became apparent. At the same time enlargement of the child's abdomen attracted the mother's attention. On examination the tip of the spleen was found at the level of the umbilicus, and the margin of the liver was palpated five centimeters below the costal arch in the midclavicular line. The bones appeared normal on x-ray study and the blood examination revealed no abnormality except a slight secondary anemia.

At the age of 18 months (November, 1933) the child had developed Mongoloid features owing chiefly to the increased prominence of the malar eminences. X-ray examination now disclosed increased trabeculation of the metacarpals, phalanges and long bones, and in the long bones marked thinning of the cortex was obvious. The inner and outer tables of the skull also appeared thin, and a marked widening of the diploe was present. The Wassermann test was negative and examination of the blood disclosed 3.89 million erythrocytes, 50 per cent hemoglobin, moderate anisocytosis, poikilocytosis and polychromatophilia, 13,500 leukocytes of which 70 per cent were lymphocytes. In addition 270 nucleated red cells per cu. mm. were present.

In March, 1934 a splenectomy was performed. Immediately prior to operation examination of the blood disclosed 3.89 million erythrocytes, 50 per cent hemoglobin; 13,500 leukocytes of which 68 per cent were lymphocytes, and 260 nucleated red cells per cu. mm. One day following a transfusion of 150 c. c. of blood the spleen was removed. A re-examination of the blood made thirteen days following the operation disclosed 4.19 million red cells, 65 per cent hemoglobin, 61,700 nucleated erythrocytes and 33,000 leukocytes of which 61,31, 8 per cent were neutrophils, lymphocytes and myelocytes, respectively.

In October, 1942 a study of the phosphorous of this patient's blood was undertaken with the cooperation of the Department of Pediatrics at Louisiana State University Medical School. The results of the one analysis we were permitted to make are recorded in table 1. The normal values shown in this table were compiled from reports in the literature.

The analysis of the patient's red cells gave abnormally high values for total and lipid phosphorous and a low value for the total esterphosphate. On fractioning the latter, evidence was found that a reduction in pyro, hexose, and glycerophosphate contributed in each instance to the decline in the total esterphosphate of the erythrocytes.

Examination of the plasma disclosed two values which were definitely abnormal, namely the low content of lipid phosphorous and the exceptionally high level of esterphosphate. The abundance of plasma esterphosphate suggests that this substance was being liberated in considerable quantity from the erythrocytes which were undergoing fragmentation and destruction.

The phosphorous values computed for the patient's whole blood on the basis of a known corpuscular volume of 25 per cent and on the analyses of the red cells and plasma indicated essentially normal levels for total and inorganic phosphorous, and abnormally high value for lipid phosphorous and a low level of esterphosphate.

During November, 1942 the patient's tonsils were removed and following this operation her condition was apparently satisfactory for approximately one month. At the conclusion of this period she became

ill while attending a picture show. Her symptoms were vague and her physician made a tentative diagnosis of mild influenza. When she entered the hospital on the sixth day of this illness she had marked dyspnea. The patient died 24 hours later at the age of 11 years. The postmortem examination was made by Dr. E. H. Lawson, pathologist at the Baptist Hospital.

At the time of necropsy her body length was 46 inches. The child was well nourished and there was no enlargement of the superficial lymph nodes. The abdomen protruded owing chiefly to enlargement of the liver, the free margin of which was at the level of the anterior superior iliac spines. The liver weighed 1735 grams. The remaining abdominal organs appeared to be normal. A few adhesions were present in the left upper quadrant and were attributed to the splenectomy that had been performed.

The heart weighed 320 grams. It was markedly enlarged owing partly to hypertrophy of the left ventricle but chiefly to dilation of the right ventricle. The valves appeared to be normal and the coronary arteries were patent at their respective origins.

The lungs were normal except for the presence of adhesions on the right side which practically obliterated the pleural cavity. There was no transudate or exudate in the pleural, pericardial or peritoneal cavity.

A lymph node and a specimen of liver were the only tissues available to us for detailed study. The histologic examination was made by Dr. J. R. Schenken, former professor and director of the Department of Pathology at Louisiana State University Medical School. His report is as follows:

*Lymph Node:* The general architecture is fairly well preserved. The peripheral sinus is patent. The lymph follicles are smaller than normal, are well formed, and show no hyperplastic germinal centers. At the periphery of the lymph node, for a distance of approximately one low-power field, there is a generalized hyperplasia of the reticulo-endothelial cells, intimately intermingled with the lymphoid tissues. All of the large reticulo-endothelial cells contain many brown-staining granules of hemosiderin pigment. Directly beneath the capsule, there is a greater concentration of reticulo-endothelial cells, each of which contains large quantities of hemosiderin pigment. In the center of the tissue, the sinuses are all dilated and filled with a pale, granular coagulum. In some cases, these spaces are at least as large as thyroid acini. Here the sinusoids are all very much enlarged and filled with closely packed hyperplastic reticulo-endothelial cells filled with hemosiderin pigment. Only small masses of lymphoid cells are present between these hyperplastic reticulo-endothelial cells.

*Diagnosis:* Hyperplasia of the reticulo-endothelial cells; lymphostasis; hemosiderosis; the hemosiderin pigment was proved to be iron-containing by the Prussian blue reaction.

*Liver:* The general architecture of the liver is preserved. The liver cords are disturbed, however, in their courses, frequently by connective tissue which emanates outward from the periportal connective tissue. In some instances, this reaches as far as the adjoining portal canal, but in others, it only spreads out for a short distance. In many cases, fragments of liver cords are caught within this scar tissue. There are no nodules of regeneration of liver cells. The liver cords themselves appear to be somewhat thinner than normal, and the sinusoidal spaces relatively larger than normal in most cases. The cytoplasms of the cells contain numerous granules of hemosiderin pigment which are uniformly dispersed throughout the cell. The nucleus contains no pigment. The Kupffer cells are increased in size and in number and also contain hemosiderin pigment, but their importance as pigment carriers is overwhelmed by the large quantities of pigment found in the liver cords themselves. In a few areas, there are foci of cells of the myeloid series which appear to be in various stages of de-

velopment and these indicate extramedullary hematopoesis.

*Diagnosis:* Hemosiderosis of the liver; early pigmentary cirrhosis of the liver.

This patient had a sister and a brother who died of Mediterranean anemia at the ages of 5 and 10 years, respectively. During infancy each of these children developed an anemia and yellowish discoloration of the skin. These changes were accompanied and followed in each instance by progressive enlargement of the abdomen, splenomegaly, marked changes involving the erythrocytes and the gradual development of Mongoloid features and extensive characteristic changes in bones. The remaining eight children in this family appear to be well. No studies have been made, however, to detect mild forms of the disease in members of the group.

SUMMARY

Fatal Mediterranean (Cooley's) anemia in three of eleven siblings born of Sicilian parents is reported. Observations on only one of these patients are presented in detail. A study of phosphorous of the blood of this child's red cells and plasma disclosed the following findings:

(a) Analysis of the erythrocytes revealed abnormally high values for the total and lipid phosphorous and low values for the total esterphosphate and each of its fractions.

(b) Analysis of the plasma disclosed a low level of lipid phosphorous and an exceptionally high level of esterphosphate.

The excess quantity of esterphosphate in the patient's plasma may have been derived from the erythrocytes which were undergoing fragmentation and destruction.

Microscopic examination of a lymph node and a specimen of liver, the only tissues available to us for detailed study, disclosed hemosiderosis, lymphostasis and hyperplasia of the reticulo-endothelial cells of the lymph node and hemosiderosis and early pigmentary cirrhosis of the liver.

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TABLE 1.

PARTITIONING OF PHOSPOROUS OF RED BLOOD CELLS AND PLASMA IN MEDITERRANEAN ANEMIA

| | Age in Yrs. | Total Phosphorous | Inorganic Phosphate | Lipid Phosphorous | Total | Esterphosphate Pyro-Phosphate | Esterphosphate Hexose-Phosphate | Esterphosphate Glycero-Phosphate |
|---|---|---|---|---|---|---|---|---|
| | | | | Red Blood Cells | | | | |
| Normal | 12 - 15 | 77.1 | 4.8[2] | 16.1[1] | 56.2[1] | 13.1[2] | 12.8[2] | 32.5[2] |
| Mediterranean Anemia | 11 | 99.5 | 4.6 | 71.6 | 23.3 | 3.4 | 7.6 | 12.3 |
| | | | | Plasma | | | | |
| Normal | 12 - 15 | 13.8[1] | 4.7[1] | 8.6[1] | 0.6[1] | | | |
| Mediterranean Anemia | 11 | 14.6 | 5.5 | 2.5 | 10.5 | | | |
| | | | | Whole Blood | | | | |
| Normal | 12 - 15 | 39.7[1] | 4.2[1] | 11.6[1] | 21.8[1] | | | |
| Mediterranean Anemia | 11 | 35.8[2] | 5.2 | 19.8[2] | 13.6[2] | | | |

1. See Stearns and Warweg.
2. See Behrendt.

# SOME PSYCHOLOGIC HAZARDS IN THE ADOPTION OF A CHILD

MILTON E. KIRKPATRICK, M.D.†
NEW ORLEANS

A physician who participates directly in arranging the adoption of an infant or child usually assumes a great responsibility. Inherent hazards in this process can and should be borne by properly constituted socio-legal agencies. In the final analysis, adoption is a function of social worker and court—with physicians, psychologists, religious groups and other interested parties acting only in an advisory capacity. The physician who oversteps his proper boundary will ultimately bring trouble to all concerned. Obstetricians and pediatricians should be particularly aware of important psychologic factors involved in the adoption procedure.

There is no need to call attention to the current prevalence of neurotic illness among both adults and children. Improper adoptions play a significant role among these. A little thought devoted to the hazards involved may prevent many avoidable psychologic sequelae. Retrospective analysis of successful and unsuccessful adoptions furnishes some knowledge of the elements that make for success or failure.

It is extremely important to establish as objectively as possible the motivating factor in any adoption. Often this is obscure and conjures up primitive and fundamental aspects of parenthood, such as the much disputed "maternal instinct." It may not be out of place here to comment on the place of children in a primitive culture—their contribution to tribal relationships. Primitive tribes in many instances did not associate the sexual act with conception or the bearing of children. Offspring were an indication that primitive deities approved the marriage or perhaps even the conduct of the tribe in general. Abundant crops also denoted the favor of the gods, consequently a lack of abundance of either crops or children was apt to be interpreted as punishment. The barren woman became an outcast. If she did not bear children, it was because the gods did not look with favor upon her. Her husband could send her back to her father and demand a refund of the purchase price. Primitive man did not look very far for an answer to his problem!

Children can be symbolic of sexuality. They signify their parents' ability to fulfill a biologic function necessary for perpetuation. How often have we heard the husband of a childless marriage referred to facetiously by his friends as being "no good." How frequently have we heard a woman say almost apologetically "We have no children." A childless marriage is a threat to perpetuity and may damage the self-esteem of the individuals involved. Physicians, psychiatrists and social workers have all encountered resultant familiar patterns: A housewife, no longer able to bear the taunts of relatives and neighbors, journeyed to a distant city and returned in a few months with an infant she successfully presented as her own. A family adopted a child and moved to a distant city, carefully severing all connections with their past life. Consider all the emotional turmoil that must underlie precautions sufficiently elaborate to insure the success of such performances!

It is unwise to advise adoption glibly, simply because one, two or more adults are lonesome and want children to brighten their lives and afford some means of entertainment; this advice is usually misguided and serves only to shake an already insecure psychologic structure. An adoption must almost certainly fail when emotional needs of adults occupy the place of paramount importance. Temporary pleasure and comfort in caring for a helpless, dependent and attractive infant may soon be replaced by the emotional upheavals centering about certain behavior disorders of childhood and later more serious problems of growing adolescents. Most physicians have seen this pattern develop; they, as well as adoptive parents, should be com-

†From the Department of Pediatrics, Tulane University School of Medicine.

pletely and continuously aware of the fact that babies grow up!

We have all seen parents—natural or adoptive—whose greatest expressed need seems to be for appreciation. "Look at all we've done for him . . . (innumerable details) . . . and he doesn't appreciate us." Expectation of tangible evidence of real gratitude or appreciation almost always leads to disappointment. Appreciation of the child by the parents determines to a large extent that child's attitude toward his parents. One of the greatest rewards and satisfactions of parenthood is honest appreciation—but such appreciation is never automatic.

Sympathy or pity alone is a poor reason for adoption. "We both felt so sorry for him. He was the kind of child your heart went out to." We recently encountered two sad results: Influential couples had visited orphanages, selected pathetic, malnourished infants and had been able to rush through adoptive proceedings, "cutting a lot of legal red tape." Later both children were found to have hereditary and familial diseases; one was a severe epileptic, the other spastic and feebleminded. Careful study and proper procedure almost surely could have avoided these needless heartaches. Combined with good judgment and other valid reasons for adopting a child, these motives may sometimes be deservedly admired. Some adoptive parents boast openly of such "poor risks", as though actually inviting or seeking admiration for their courage. Pity is an unworthy motive for adoption; there is a vast difference between love and sympathy.

No adoption is likely to succeed unless both parents are committed to it. All too often we find the mother to be the only party genuinely interested, the husband exhibiting a tolerant or passive attitude: "Let the wife have her way." Many women have the mistaken notion that a child in the house may reform a wayward husband. On the contrary, such an addition usually only creates new problems—and consider the rejection to which the child may be exposed when such a plan fails. On the other hand, some happily married though childless couples honestly feel that a child in the home would make their happiness just a little more complete; an unfortunate result of adoption in such an apparently benign situation has recently come to our attention. A father's tremendous attachment to a ten-year-old girl adopted in infancy has led to jealousy and feelings of guilt on the part of the mother. The little girl has unwittingly come between this couple and brought their marriage close to disaster.

Five years ago, a young couple, on advice of their family physician, took a newborn infant who had been abandoned by an unmarried mother. Caring for this baby became the most satisfying experience in their lives. A legal adoption was never completed, however, as the mother never signed a release. Now, with improved fortune and a successful marriage to an understanding husband, the real mother demands return of her child, and legal counsel finds no alternative. The consequences are not difficult to imagine, and the responsibility rests quite squarely on the physician's hasty attempt to find a solution to an unpleasant situation.

Regardless of how much information a physician may have concerning the child or prospective adoptive parents, he should never specifically recommend adoption. The risks are too great, requiring almost foolhardy courage. After careful investigation and evaluation conducted objectively by impartial workers—and always including a trial period of placement—he should merely point out the assets and liabilities involved. The responsibility then lies solely with the court and the adoptive parents.

Infants are often adopted so early or so hastily that no adequate study of background, physical condition, mental status, or estimate of future capability can be made. Many sad results of this unfortunate error are brought to our attention each year. Exclusion of congenital syphilis by appropriate means should be insisted upon in every case. Of even greater importance, though not as quickly accomplished, are studies justifying more than a casual eval-

uation of developmental or mental status. No effort should be spared to exclude objectively uncorrectible disease of mind or body and to ascertain that the child is educable at least to the level of eventual self-support. We then may be able to avoid the tensions and frustrations in the adoptive home where parents have intended to give the child a college education only to find, after repeated school failures, that his intelligence is limited to seventh grade achievement.

A physician should not advise or actively participate in arranging adoption of a child. If the physician has knowledge of a child apparently suitable for adoption, he should direct the mother to the proper social agency where she may execute a release. If he knows of a family who wish to adopt a child, he should refer them to this same agency. Complete information regarding agencies doing adoption work may be obtained through the Community Chest in our larger cities. In rural areas the State Department of Child Welfare should be contacted. Copies of the laws pertaining to adoptions may be obtained through that agency.

This subject of adoptions has assumed such importance in recent years that the Editor of the Journal of the American Medical Association took occasion to comment on it recently in the editorial columns as follows:

"The control of illicit traffic in babies requires that all adoptions should be a matter of public concern. The state, in taking control, has developed certain general principles which have been republicized by the United States Children's Bureau. The general principles which should govern adoption are set forth by the bureau as follows:

"1. Adoption proceedings should be held before a local court accustomed to handling children's cases; petitioners for adoption should reside and be known locally.

"2. The court should have the assistance of the state welfare department, which should be made a party to every adoption proceeding.

"3. Consent to adoption should be obtained from natural parents or other persons or agency having legal responsibility.

"4. Court hearings should be closed and records confidential.

"5. A trial period of residence, preferably for one year in the adopted home, should be required prior to issuance of the final adoption decree.

"6. Provision for removal of children from unsuitable homes should be made as well as provision for their care and guardianship after such removal.

"7. All applicable statutes should assure the welfare of the child and the rights and obligations of the parents."

There is probably nothing in the American way of life more universally satisfying than a happy home where children are loved and appreciated. That children are impossible in some marriages is not always an irremediable misfortune. Many intelligent couples have found tremendous satisfaction and happiness in taking into their little family group a child of a union which has failed to provide him not only with the securities of childhood but with the love, acceptance and tolerance so essential for the development of a healthy adult personality.

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## HYPERTROPHIC PYLORIC STENOSIS

### (ANALYSIS OF THIRTY-SEVEN CASES AT CHARITY HOSPITAL)

L. J. O'NEIL, M. D.†

NEW ORLEANS

The purpose of this paper is to report recent changes in the mortality rate observed at Charity Hospital for congenital hypertrophic pyloric stenosis and to consider some of the factors responsible for these changes.

In a study covering the five year period which immediately preceded the years considered in this communicatian (1939 to 1945) Romano and McFetridge[1] reported a mortality of 34.4 per cent in a group of 32 infants with hypertrophic pyloric stenosis. Fourteen of these patients were treated

†From the Department of Surgery, Louisiana State University School of Medicine, and the Charity Hospital of New Orleans, Louisiana.

medically with six deaths and 18 were treated surgically with five deaths. Romano and McFetridge were inclined to attribute this excessive mortality to the prolonged medical treatment undertaken prior to surgery.

Between January, 1939 and October, 1945, the 36 infants with whom this paper deals were operated upon at Charity Hospital for hypertrophic pyloric stenosis. The results were as follows:

| | Cases | Deaths | Mortality |
|---|---|---|---|
| 1939 | 3 | 1 | 33.3 |
| 1940 | 4 | 2 | 50.0 |
| 1941 | 10 | 1 | 10. |
| 1942 | 6 | 0 | 0. |
| 1943 | 4 | 0 | 0. |
| 1944 | 6 | 0 | 0. |
| 1945 | 3 | 0 | 0. |
| | 36 | 4 | 10.8 |

The infant who died in 1939 had been given normal saline and glucose solution by hypodermoclysis for 19 days preoperatively. He expired 36 hours after operation and at necropsy the death was attributed to pyloric stenosis and marasmus.

During 1940 there were two deaths, one of which resulted from bronchopneumonia on the fourteenth postoperative day and the other from mesenteric thrombosis on the twenty-fifth day following operation.

The death in 1941 was due to generalized peritonitis secondary to penetration of the duodenal mucosa at the time of operation. This death occurred on the fourth postoperative day and it is the only fatality directly traceable to opening the mucosa of the duodenum. This accident occurred in 17 of the 36 cases reported in this paper. In each instance the perforation was closed and an omental graft was sutured over the site.

Between January, 1942 and October, 1945, 19 infants have been operated upon for hypertrophic pyloric stenosis without a death. During this period the preoperative period of preparation averaged four days, whereas prior to 1941 it averaged three weeks. This recent decline in the mortality rate for infants with hypertrophic pyloric stenosis observed at Charity Hospital is not due to the technical skill of any one surgeon. The patients reported in this paper were operated on by twenty different operators most of whom were members of the resident staff of the hospital.

Attention is again called to the fact that since January, 1942, 19 infants with hypertrophic pyloric stenosis have been operated upon at Charity Hospital with no mortality. This splendid result is attributed first to the preoperative care administered by the pediatric staff and second to prompt rather than delayed operation.

### CONCLUSION

**The recent decline in the mortality rate for congenital hypertrophic pyloric stenosis at Charity Hospital is attributed to efficient preoperative preparation followed by prompt surgical relief of the condition.**

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## CLINICO-PATHOLOGIC CONFERENCE

### ACUTE LARYNGOTRACHEOBRONCHITIS VS. DIPHTHERIA*

WALLACE SAKO, M. D.†
and
JOHN R. SCHENKEN, M. D.†

NEW ORLEANS

A white female, aged 4 years, was admitted February 7, 1945 and died February 10, 1945.

*Chief Complaint*: Difficulty in breathing.

*Present Illness*: On February 5, 1945 the patient awoke with hoarseness of voice. The mother believed that the child had a cold and treated her with nose drops and cough syrup. The next day the patient began to cough and complain of sore throat. On February 7, 1945 a fever of 101° F. developed and the child began to have considerable difficulty in breathing which became progressively worse until admission to Charity Hospital.

*Past History*: The child enjoyed good health except for the usual colds, mild diarrhea, and an attack of measles in 1944. There was no history

†From the Departments of Pediatrics and Pathology, Louisiana State University School of Medicine and the Charity Hospital of Louisaina at New Orleans.

of immunizations and contact with diphtheria or other contagious diseases.

*System Review*: Negative.

*Physical Examination*: Temperature 103.6° F. rectally, pulse 150 per minute, respiration 32 per minute.

The patient was a well developed, well nourished, white female child of 4 years lying in bed and having difficulty in breathing. The tonsils were moderately enlarged and the pharynx was red and inflamed but no membrane was visible. The lungs, according to the interne, were clear to auscultation and percussion. The next day, however, upon examination the chest revealed on auscultation loud sonorous rhonchi with fine crackling and coarse mucoid rales throughout both lung fields. The heart was normal except for the rapid rate. The abdomen, extremities, genitalia, and nervous system were negative.

*Laboratory Data*: Nose and throat cultures taken on admission revealed no diphtheria organisms but was positive for alpha streptococci. There was a polymorphonuclear leukocytosis. The urine, Kline and Kolmer, and Mantoux tests were negative. The Schick test was positive.

*Course in Hospital*: There was marked dyspnea with rapid feeble respirations and slight tugging of the infra- and suprasternal notches. An emergency tracheotomy was performed shortly after admission. The patient was given sodium sulfadiazine intramuscularly grains 1 1/2 per pound of body weight initially, followed by a maintenance dose of grains 1 1/2 per pound per day in six equal doses every four hours. Diphtheritic antitoxin 10,000 units was given intramuscularly. The next day an additional 50,000 units of diphtheritic antitoxin was given intramuscularly. On February 9, 1945 the respiratory distress became progressively worse. The patient was bronchoscoped and mucopurulent material was suctioned out. The bronchi appeared inflamed and edematous. Following bronchoscopic suction, the respirations improved slightly. The lungs revealed sonorous and mucoid rales. Penicillin was then started 20,000 units and 10,000 units intramuscularly every three hours. At 12:30 a. m. on February 10, 1945 the patient was breathing feebly and quietly. There was no cyanosis. The child was very restless, so phenobarbital was prescribed for sedation. At 4:30 a. m. the patient suddenly ceased breathing without struggling. An emergency bronchoscopy was done. The trachea was very edematous and was covered with thick plastic exudate which could not be removed. The patient expired at 4:50 a. m.

CLINICAL DISCUSSION

This is a 4 year old child whose history has been recorded above. The illness began with hoarseness which would point to laryngeal involvement. Fever initially was 101° F., then rose to 103° F., and finally to 104° F. There was marked and progressive respiratory embarrassment. Hoarseness, fever, and dyspnea suggest three conditions commonly encountered in a child of this age: (1) simple croup, (2) diphtheria, and (3) acute laryngotracheobronchitis.

1. *Simple Croup:* In this condition there are slight inflammation and spasm of the larynx giving rise to dyspnea and respiratory stridor. It commonly occurs in children between the ages of six months to five years. Characteristically the child appears perfectly well during the day except for slight hoarseness and cough. In the evening the hoarseness, barking cough, and dyspnea make their appearance and become progressively worse until midnight when dyspnea, cyanosis, and loud respiratory stridor with retraction of the infra- and suprasternal fossa and intercostal and supraclavicular spaces make their appearance. The child at this time is extremely restless, apprehensive, anxious and prostrated. The respirations are slow and labored, the cough loud and barking, the pulse rapid, and the temperature normal or slightly elevated up to 101° F. When daylight comes the symptoms gradually subside and the child lapses into a restful sleep. All during the day the child remains well except for slight cough and hoarseness. When nightfall comes the process repeats itself again and lasts for three to five days. Recurrent attacks may occur every few weeks or so but the prognosis is always good.

In this case, high fever up to 104° F., progressive non-spasmodic dyspnea without diurnal variations, lack of history of previous attacks, marked evidence of inflammation of the larynx, lack of response to ordinary measures, and the fatal termination would rule out the possibility of simple croup.

2. *Laryngeal diphtheria:* In a child of 4 years, fever and laryngeal involvement would necessitate a provisional diagnosis of diphtheria. No membrane was seen either in the pharynx on admission or in the larynx the next day. The pharynx was only slightly inflamed but the larynx was markedly

injected and edematous. This would tend to rule out diphtheria, although it might be possible that the membrane may not have been visualized due to severe edema. The majority of the cases of laryngeal diphtheria (75 per cent) is a direct extension from the pharyngeal involvement, and only about 25 per cent of pharyngeal diphtheria involves the larynx. Approximately 12½ per cent of diphtheria is purely laryngeal in character. A bronchoscopic examination following tracheotomy revealed edema and inflammation with mucopurulent exudate but no membrane. Since it is highly unlikely for diphtheria of five days' duration not to form a membrane, we should consider something else than diphtheria.

A temperature of 103-104° F. is higher than expected for diphtheria. A pulse rate of 150 per minute and extreme prostration are consistent with diphtheria. However, the patient did have a tremendous amount of dyspnea, which in a child of this age will cause the pulse rate to rise, and, consequently, we cannot say from the rapid pulse rate that the condition is diphtheria. The fact that the patient had received no immunization and the Schick test was positive are both highly significant. In reviewing all the cases of diphtheria admitted to Charity Hospital during the years 1941-1945, it was found that there has been no clinical case of diphtheria occurring in children who had been completely immunized against the disease. The fact that the patient did not come in contact with a diphtheria case is of no significance whatsoever.

Unfortunately direct smears were not done. The fact that one culture for diphtheria was negative does not mean much. The presence of diphtheria bacilli on culture gives a positive diagnosis; a negative culture does not rule out diphtheria. Treatment with antitoxin was justified in spite of the negative culture. Let us remember the admonition that any condition requiring a diphtheria culture justifies treatment with antitoxin.

The 10,000 units of diphtheria antitoxin administered intramuscularly was grossly inadequate. This is borne out by the fact that the interne was uneasy the next day and administered an additional 50,000 units intramuscularly. Divided dosage such as this is not as effective as a single adequate dose. All severe laryngeal cases should receive maximum dosage, possibly 100,000 units. Some clinicians say that pure laryngeal diphtheria requires a small dose since toxicity and absorption are not as great as the pharyngo-laryngeal form. This is not always true in my experience. At any rate, I would not take any chances.

The intramuscular route of administration should not be used in emergencies such as this. The absorption is too slow. The intravenous route should have been used for rapid effect. Response, then, should be apparent in six to 12 hours. By the intramuscular route, maximum absorption occurs in 24 to 48 hours. This is too slow in obstructive diphtheria cases. The subcutaneous route should never be used for therapy since maximum absorption occurs in 72 to 96 hours. I would have given this case 100,000 units of diphtheria antitoxin, 50,000 units intravenously diluted with at least an equal volume of normal saline or glucose solution and 5 minims of 1:1000 adrenalin solution and 50,000 units intramuscularly or all intravenously. Of course, sensitivity tests for horse serum should be performed and due precaution should be exercised in the administration of any heterologous serum.

3. *Acute laryngotracheobronchitis:* A temperature of 103-104° F. is more consistent with the diagnosis of acute laryngotracheobronchitis than of diphtheria. The interne who examined the child stated that the lungs were clear to auscultation and negative to percussion. When we examined the child, however, the respiration was so noisy and snoring that it tended to obscure the rales which were heard over both lung fields. The heart rate was very rapid as can be expected. The abdomen was negative. Upon close examination cyanosis was present although this was not noted by the interne. Dyspnea and toxemia were so marked that prostration and exhaustion were noticeable. The tremendous exudate

in the tracheobronchial tree plus marked exhaustion caused the respiration to become shallower and shallower so that under oxygen the respiratory excursions were scarcely noticeable. The anoxic condition made the child extremely restless. Apparently the final exitus with cessation of respiration was not a sudden process but a gradual one of drowning from the mucopurulent exudate in the bronchi and trachea. Consequently, no struggling occurred at the time of death.

Emergency tracheotomy was performed and rightly so. It is not always an easy problem to decide as to when tracheotomy is indicated. In pure laryngeal obstruction with marked retraction of the supra- and infrasternal notch the indications are clear cut and marked and prompt relief from dyspnea is obtained. In cases of dyspnea wherein the obstruction is not in the larynx but in the tracheobronchial tree, tracheotomy gives no relief. Other cases, such as the patient above, present obstructions in the larynx, trachea and bronchi and it is in these cases that decision for surgical intervention is difficult. The larynx is edematous and the trachea and bronchi are filled with a mucopurulent exudate. Tracheotomy may give no relief. Oftentimes the strain of subjecting the patient to surgery will in itself be fatal. Therefore, in dyspnea without a tug on the xyphoid, tracheotomy as a rule should not be done, and the patient should be handled conservatively. Since the primary difficulty is caused by the thick mucoid, or purulent exudate, from the theoretical standpoint, the ideal treatment would be some agent which can liquefy this exudate to permit the patient to cough it up or to permit absorption to occur. We do know that steam, carbon dioxide, and expectorants such as ammonium chloride and ipecac tend to liquefy exudates, whereas oxygen, heat, and atropine tend to thicken exudates in the tracheobronchial tree.

In the presence of dyspnea and cyanosis, it is difficult to decide whether oxygen, carbon dioxide, or steam is indicated. Carbon dioxide 5 to 10 per cent with oxygen 90-95 per cent has a dehydrating effect. Pure carbon dioxide has a liquefying effect but is dangerous to use for any length of time. It is impractical to combine steam and oxygen. The efficacy of a croup tent or oxygen tent can be determined only by trial; whichever makes the patient more comfortable and gives relief should be used.

Some cases of acute laryngotracheobronchitis are due to diphtheria but the majority are due to the streptococcus, pneumococcus, influenzal bacillus, and the staphylococcus or a mixture of these organisms. In our case a culture was not taken during life, so the etiologic agent was not known. Modern treatment offers a much better outlook for acute laryngotracheobronchitis. Sulfonamide therapy is successful in some instances, but at best the response is slow probably because of the thick muco-fibrinopurulent exudate in the presence of which the sulfonamide drugs will not often act due possibly to inactivation by para-aminobenzoic acid. Penicillin would be the drug of choice except in the influenzal type. In this case, sulfonamide was given in adequate dosage but penicillin was started late and was not given an adequate trial. If the etiologic organism is the influenzal bacillus, sulfadiazine and type specific anti - influenzal rabbit serum should be given. Alexander, of New York City, reports good results with the use of type B influenzal rabbit serum. The antiserum should be given in doses of four ampoules (100 mg.), two ampoules intravenously and two ampoules intramuscularly. If no improvement occurs in 24 hours, the dose should be repeated. If the etiologic organism is the type B influenzal bacillus response occurs in 48 hours.

Acute non-diphtheritic laryngotracheobronchitis is a serious disease, and in the severe form carries a high mortality. From 1936 to 1941, 140 such cases were treated in Charity Hospital with a mortality of 51 per cent. Most of these cases have had an emergency tracheotomy. In the pre-sulfonamide era, patients with acute laryngotracheobronchitis subjected to tracheotomy usually died either from the primary disease or from secondary infection with other organisms, usually the hemolytic streptococcus. To prevent secondary complications, all

tracheotomized patients should receive sulfonamide and/or penicillin.

From the foregoing discussion, therefore, I believe that the diagnosis in this case is acute laryngotracheobronchitis probably due to the streptococcus. The high temperature, absence of membrane, and profuse mucopurulent exudate in the tracheobronchial tree point more strongly to the above diagnosis than diphtheria.

*Clinical diagnosis:* Acute laryngotracheobronchitis probably streptococcal.

PATHOLOGIC DISCUSSION

This is a well nourished and well developed child of 4 years who showed a considerable amount of rigor mortis which is what you would expect in an acute disease of this type. There was no discharge from the ears and nose. The lips were bluish. In the mid-line over the trachea, there is a recently made aperture that was obviously the tracheotomy wound which went into the trachea. The aperture is about one centimeter in diameter and contains a thick, greenish-yellow exudate. The fingernails, curiously, did not show any cyanosis. The peritoneal cavity showed nothing of importance. The pleural and pericardial cavities were both free of fluid and exudate. The heart was normal and weighed 65 grams. In a child 4 years of age the heart should weigh approximately 73 grams, so this heart can be considered as within normal range.

*Lungs.* The right lung weighed 115 grams and the left lung 105 grams. Their normal weights in a child of this age are 90 grams and 85 grams respectively so both lungs are somewhat increased in weight. The lungs contained air throughout except for some dependent areas of purplish discoloration. Frothy fluid exuded from the cut surfaces but there were no areas of granularity present. In other words, it was not the impression of the pathologist that pneumonia was actually present at the time the child died. The lower trachea and bronchi, although this is not visible on the slides, (which I will show you in a moment) were completely occluded with an extremely thick, tenacious, greenish-yellow exudate which was firmly adherent to the wall, just exactly as the clinician predicted. There was no membrane present *per se*—no membrane which looked like diphtheritic membrane. The mucosa was fiery red so the primary inflammatory reaction seems to have been in the trachea and larger bronchi.

*Larynx.* The larynx was injected and slightly edematous but no membrane was present.

*Spleen.* The spleen weighed 70 grams. The normal weight should have been 40 grams. So we have definite enlargement with very prominent Malpighian corpuscles and softening of the pulp as we would expect to see in severe toxic splenitis. In severe toxic splenitis in children, the Malpighian corpuscles become hyperplastic in a matter of days.

*Liver.* The liver weighed 950 grams in contrast to the normal weight of 516 grams at this age. It shows definite enlargement and the cut surface shows a preservation of the markings, a reddish-brown color of the parenchyma, and a group of bright yellowish nodules, pinhead in size, surrounded by a hyperemic zone.

*Pancreas, adrenals, gastrointestinal tract, genito-urinary organs.* These showed no important changes.

I will now show you some pictures of the organs. This is a dissected specimen showing the tongue, tonsils, epiglottis, larynx, trachea, and bronchi. The first thing that strikes you is the extreme redness of the mucosa of the trachea and bronchi. It is fiery red beginning way up in the epiglottis and ending in the larger bronchi. The other finding which is of great importance and interest was the exudate that was present. This is not so well seen now because it is glistening, somewhat translucent, and has sloughed off, but here and there you can still see remnants of this exudate. When the trachea and bronchi were first opened, they were completely occluded by this exudate. Here is a posterior view of the tracheotomy wound. The exudate present on the mucous membrane is clearly visible on this close-up view.

This is a section of the spleen. The pinkish gray mottled appearance is due to the hyperplastic Malpighian follicles. You will also notice that there is a red rim of homogeneous tissue all around the spleen. This

is rather commonly seen. It is due to the fact that when the spleen is removed and is exposed to the air for a period of time, the blood at the periphery of the spleen becomes redder because of oxidation.

This is a cross-section of the trachea. It is an amazing section. Here are the tracheal rings and here is the lining of the trachea. Some of the submucosal tags are still present but there is complete absence of the mucous membrane and on the surface there is attached a very thick mucoid exudate which contains a relatively small number of inflammatory cells. This coagulation necrosis of the mucous membrane is a remarkable thing. There is practically no mucosa left except for some little ducts which we see going down to the mucous secreting glands. It differs from a diphtheritic membrane in that we see practically no fibrin. I would never have dreamed from the gross appearance that the mucous membrane would show so much necrosis.

This is a section of the tonsil which shows a normal appearance. Here is a section of the spleen which shows the large follicles and a large amount of edema, red blood cells, and polymorphonuclear leukocytes in the pulp. This is what you would expect to find with severe septic splenitis and accounts for the splenic enlargement. The section of the heart shows no abnormalities. The microscopic section of the lung shows that 75 per cent of the air containing spaces are filled with edema fluid with very few inflammatory cells. The increase in the number of polymorphonuclear cells in the alveolar wall shown here would add to this patient's distress. This is interstitial pneumonia which occurs quite frequently in children and oftentimes is the result of a blood stream infection. In this case we were unable to prove a blood stream infection because the postmortem blood culture was sterile.

The section of the liver shows a healing caseous focus surrounded by a zone of epithelioid cells. Obviously this is a lesion resulting from primary dissemination of the first infection type of tuberculosis. It is not miliary tuberculosis in the true sense since miliary tuberculosis is a clinical entity with progressive inoculations of numerous tubercle bacilli into a blood stream. In primary tuberculosis infection the bacilli lodge in various organs and then recede. The primary focus must be in the lung but we missed the boat somewhere since I cannot find it.

Postmortem culture from the trachea revealed Aerobacter, *Staphylococcus aureus*, and alpha streptococcus. From an etiologic standpoint these organisms are not significant and not very helpful in diagnosis. We know perfectly well that the alpha streptococcus can be routinely cultured from the nose and throat of normal individuals. Everybody harbors this streptococcus at all times, and this child may have been a carrier of this organism which might not have been etiologic.

Two mistakes were made here. In the first place a postmortem culture for diphtheria was not made because it was assumed that diphtheria was ruled out on clinical grounds and from the antemortem culture. The second mistake was that a direct smear of the exudate was not submitted with the culture. The reason for the primary or direct smear is this: suppose you have a mixed flora with some rapidly growing organisms in them as well as some delicately growing organisms, such as some strains of streptococci, the rapidly growing organisms would overgrow the more delicate organisms very quickly and as a result you would overlook the tiny little colonies in the culture plate. However, if you see a large number of chains of cocci on the primary smear then you would be certain that you are dealing with a streptococcus and you would look harder for the tiny colonies between the larger more luxuriant colonies. On section the bacteriologist found several groups of bacteria which look like streptococci. These streptococci may be etiologic:

The pathologic diagnosis is:

1. Acute laryngotracheobronchitis probably streptococcal.
2. Acute interstitial pneumonia—terminal.
3. Septic splenitis.
4. Fatty changes in liver, with edema.
5. Primary tuberculosis.

NEW ORLEANS
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*Established 1844*

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THE JOURNAL *does not hold itself responsible for statements made by any contributor.*

## THE PEDIATRIC NUMBER

This is the first of a proposed series of December issues of this journal devoted exclusively to pediatric topics. Included in the present number are contributions originating from activities in the Departments of Pediatrics of Tulane and Louisiana State University medical schools. We hope that articles for later issues will be submitted by many other physicians in this field. The publication of "specialty numbers" is not a new editorial policy but serves a useful purpose both to stimulate interest in the various specialties and to organize related material.

The wide variety of problems in this issue should be of interest to all practitioners who deal with children. Uyeda's article stresses once more the importance of rheumatic infections in the South; these obviously are much more frequent than commonly believed. Cummins' discussion of genetic patterns should command wide interest. There are many lessons to be learned from the clinical pathological conference presented by Sako and Schenken. Early recognition and prompt treatment of hemolytic disease of the newborn is gratifying; Dr. Aleman reports three additional cases observed recently. Closer cooperation of pediatrician and surgeon is undoubtedly the major factor contributing to improved results in treatment of pyloric stenosis at Charity Hospital, as pointed out by O'Neil. Dr. Hansen's tables for dietary evaluation will be appreciated by many. A clear-cut statement of the doctor's role in adoption proceedings has been furnished by Kirkpatrick. Phillips and Stewart analyze the characteristic features of annular areas of pulmonary rarefaction observed in children convalescing from pneumonia. Williamson and Crawford have summarized available information concerning erythroblastic (Cooley's) anemia and reported their studies in a child observed over a period of several years.

It has been a great pleasure to prepare this "specialty issue", and it is hoped that physicians dealing with diseases of children will now find time to contribute suitable articles very soon for inclusion in the second number planned for next December.

R. V. PLATOU, M. D., and
C. A. STEWART, M. D.†

## ERYTHROBLASTOSIS FETALIS

Recent articles on erythroblastosis fetalis published in medical and lay journals emphasize the frequency of this condition and

†From the Departments of Pediatrics, Tulane University School of Medicine, and Louisiana State University School of Medicine.

its gravity. Scientific discussions of the relationship of the Rh factor and its subdivisions to this disease are provided by the splendid treatises of Levine, Weiner, Race, Taylor, Davenport, and other investigators. This occasion is taken to stress only a few of the practical aspects of the problem the disease presents.

Owing to the fact that the offspring particularly of an Rh positive father and an Rh negative mother may have erythroblastosis fetalis, all parents should have their respective Rh factor determined before their infant is delivered. Thus, prior to the birth of the child information of great value can be obtained relative to the probability that the expected infant may develop erythroblastosis fetalis, especially when the mother is a multipara. Furthermore, since a very minute quantity of Rh positive blood administered by any route to an Rh negative female may render the recipient liable for years to have offspring with erythroblastosis fetalis, the necessity for determining the Rh status of the donor each time blood is given to an Rh negative female infant, child, or adult is obvious. This recent advance enables physicians to avoid a previously unsuspected danger that lurks in intramuscular and intravenous injections of blood, which prior to the discovery of the Rh factor, was considered to be entirely satisfactory. We have undoubtedly entered the era when it is imperative that Rh negative donors be used no matter how little, by what route, or at what age in life blood is given to an Rh negative female patient.

It is now known that the Rh factor is a complex antigen possessed of many subfactors, and on this basis the occasional occurrence of erythroblastosis fetalis, when the mother and father as well as their infant are Rh positive, is explained. In spite of the existence of these subfactors the present routine method of determining the Rh factor enables the physician to avoid inducing harmful immunizations in perhaps one hundred per cent of the cases provided the precautions mentioned above are exercised when blood is administered to female patients.

SUZANNE SCHAEFER, M. D.†

†From the Department of Pediatrics, Louisiana State University School of Medicine.

# ORGANIZATION SECTION

**The Executive Committee dedicates this page to the members of the Louisiana State Medical Society, feeling that a proper discussion of salient issues will contribute to the understanding and fortification of our Society.**

***An informed profession should be a wise one.***

## MEDICAL TRENDS IN WASHINGTON

There was held in Chicago on October 19 and 20 at the headquarters of the A.M.A. a conference of the Council on Medical Service and Public Relations. As a result of this conference the Council, in keeping with resolutions passed, has called another meeting for the purpose of discussing specifically voluntary prepayment medical insurance plans now in operation in the United States with the object of molding some form of national insurance program which could be utilized by all the states.

The Chairman of our Committee on Prepayment Medical Insurance, Dr. O. B. Owens, of Alexandria, is going to represent our State Society at this meeting. Unquestionably he will be able to bring back to our organization and to his committee, valuable information and proposals along the lines as indicated above. This meeting was no doubt called just prior to the meeting of the House of Delegates of the A.M.A. as it was felt that this was an opportune time to prepare recommendations to the House of Delegates for their consideration. We should all be greatly concerned and

watch with a great deal of interest what develops at this conference as well as the action taken by the House of Delegates which will certainly indicate the trend of thought on this vital subject.

You have probably noticed in the November 20 issue of the newspapers a statement released by Associated Press giving the five-point program submitted by President Truman as legislation which should be enacted by the present Congress. The first one of these proposals is a compulsory national insurance health program along the lines mentioned in the Wagner Bill, taxing the employer and employee for such service. Unquestionably it is planned to amend the present Social Security Act because immediately after the President presented this program Senators Wagner and Murray introduced a bill in the Senate and Representative Dingell introduced a new bill in the House, containing provisions for the plan as outlined. We do not know the exact form in which this legislation has been presented but we are taking the opportunity of writing to our senators and representatives to obtain a copy of these bills as soon as they are printed. Can the leopard spots be so easily changed? Our various component societies and individual physicians should also secure copies of these bills and familiarize themselves with this proposed legislation. Just as soon as we can get any new data you will be apprised of this through the Journal or otherwise.

You should also be acquainted with the fact that recently the Senate Committee on Education and Labor, through a subcommittee, introduced a completely revised Senate Bill 191, known as the Hill-Burton Hospital Construction Bill. This was acted upon favorably by the committee and is now on the floor of the Senate. The new bill, in comparison with the original bill, is highly favorable to the medical profession. Section 635, the last paragraph of the bill, reads as follows: "Except as otherwise specifically provided, nothing in this title shall be construed as conferring on any Federal officer or employee the right to exercise any supervision or control over the administration, personnel, maintenance, or operation of any hospital with respect to which any funds have been or may be expended under this title." When this report was submitted to the Senate, Senator Murray, Chairman of the Committee, submitted a minority report. He contended that the revised bill removed entirely Federal control and he thought this was objectionable due to the fact that the Federal government would finance the projects mentioned in the bill. He feels too much jurisdiction has been conferred on the state and too much authority in a Federal Hospital Council which is provided for in the bill. On the whole he seems to think that the Wagner-Murray Bill, Senate Bill 1050, would be much more appropriate and contains all the essential needs for hospital expansion.

In view of the recent developments from the White House and the favorable action taken on Senate Bill 191, it will be very interesting to watch the trend of legislative thought in trying to erase the differences which have developed by leaders in Congress.

Another very important bill of serious concern to the medical profession is the Pepper Bill, Senate Bill 1318, which calls for a continuance of the Emergency Maternity and Infant Care program. Your attention is particularly called again to this measure as in our opinion this is one of the most vicious and destructive pieces of legislation that the medical profession has ever encountered. While the original EMIC Program was a wartime directive from the Children's Bureau, it may now become a legislative enactment. This vicious legislation proposes to extend maternity care to every mother and care for every child up to twenty-one years of age. The funds for payment of such service to doctors will be paid by the Federal government through the Children's Bureau. Under such a plan it would not be long before the entire practice of medicine would be supervised, administered and controlled by the Federal government. All doctors who have not read this bill should write their congressmen for

a copy and after reviewing send them opinions concerning same.

### 1946 ANNUAL MEETING

Many of our members over the state are asking about plans for our 1946 annual meeting. The Executive Committee selected the dates of April 29, 30 and May 1 for the meeting on the presumption that a three-day meeting would be held. This was predicated on the possibility of holding a scientific program and whether the hotels in the various localities could accommodate the meeting. Although we have sent inquiries to the various centers where the meeting might possibly be held with the hope that arrangements could be made and an invitation extended for the meeting, we have not yet been honored by such an offer. Those who have responded have found it almost impossible for the hotels in their particular locality to take care of the attending physicians or furnish other facilities required for the meeting. There seems to be a great interest by the doctors of the state for an annual meeting and every effort is being made to make the necessary arrangements. However the prospects are not very encouraging for a three-day meeting including a scientific program and a meeting of the House of Delegates.

### DUES FOR 1946

Your attention is called to the fact that dues for 1946 membership in the State Society ($25.00) should be paid in December of this year by all active members. Doctors should communicate at once with the secretaries of their local societies and get their dues in as early as possible. The State Society will still delete the dues for those doctors who are continued in military service. Deletion for doctors discharged from military service will be effective for the current year in which terminal leave expires although they may have been discharged before the first of that year.

# LOUISIANA STATE MEDICAL SOCIETY NEWS

## CALENDAR

### PARISH AND DISTRICT MEDICAL SOCIETY MEETINGS

| Society | Date | Place |
|---|---|---|
| East Baton Rouge | Second Wednesday of every month | Baton Rouge |
| Morehouse | Second Tuesday of every month | Bastrop |
| Orleans | Second Monday of every month | New Orleans |
| Ouachita | First Thursday of very month | Monroe |
| Rapides | First Monday of every month | Alexandria |
| Sabine | First Wednesday of every month | |
| Second District | Third Thursday of every month | |
| Shreveport | First Tuesday of every month | Shreveport |
| Vernon | First Thursday of every month | |

### TOURO INFIRMARY

The regular meeting of the Medical Staff of Touro Infirmary was held on November 14, 1945, at 8 p. m. The Department of Pathology conducted a clinico-pathologic conference with a discussion by Dr. Thomas Findley. Dr. James W. Burks, Jr., spoke on "Coccidioidal Granuloma" and Dr. L. F. Klenke on "Estimation of Surgical Risk of Patients and the Value of Premedication in the Administration of an Anesthetic."

### OCHSNER CLINIC

A meeting of the Ochsner Clinic staff members and fellows was held on Friday, November 23 at 8:30 p. m., in the reception room of the Clinic. Dr. Edgar Burns spoke on "Complications of Congenital Anomalies of the Urinary Tract"; Dr. Paul T. DeCamp spoke on "Congenital Cyst of the Lung" which was discussed by Drs. R. V. Platou and Julius L. Wilson. Dr. Robert P. Howell talked on "Enterocoele with Abscess of the Ovary", discussed by Drs. Curtis Tyrone and Conrad Collins.

### CHARITY HOSPITAL

A meeting of the Medical Division of the Charity Hospital Visiting Staff was held on Tuesday, November 20 at 8 p. m., in the auditorium of the hospital. The program consisted of the following:

"Histoplasmosis" and "Calcified Intra-abdominal Tumor" by Dr. D. J. Seabury. Dr. Louis Levy spoke on "Bacterial Endocarditis" which was discussed by Dr. Edgar Hull.

### SOUTHERN BAPTIST HOSPITAL

A meeting of the Clinical Staff of the Southern Baptist Hospital was held on November 27 at 8 p. m. The program was as follows: "Some Reparative Problems in Military Surgery" by Dr. John F. Oakley; "Handling of Casualties on a Hospital Ship" by Dr. E. Garland Walls; motion picture "Skin Grafting of War Wounds and Observations on Wound Healing" by Dr. M. Converse, discussed by Dr. Waldemar R. Metz; "Death Report" by Dr. Philip M. Tiller, Jr.

### HULL HONORED

Dr. Edgar Hull, head of the Department of Medicine at Louisiana State University Medical School, was selected by the graduating class as the physician who has done "most for the welfare of the school and most for the student body during the past school year."

Dr. Lewis E. Jarrett, director of Touro Infirmary, represented Louisiana hospitals at a meeting of the American Hospital Association in Chicago.

### GENERAL KIRK RECEIVES DISTINGUISHED SERVICE MEDAL

Major General Norman T. Kirk, Surgeon General of the Army, has been awarded the Distinguished Service Medal by General Brehan Somervell, Commanding General of the Army Service Forces, in recognition of his "outstanding leadership . . . in directing the largest Medical Department in the history of the United States Army."

### GRANT FOR THE STUDY OF HYPERTENSION

The Winthrop Chemical Company has made a grant of $2500.00 to the Alton Ochsner Medical Foundation for research in hypertension. The chemical phases of this problem are directed by Otto Schales, D.Sc.

### MEDICAL WOMEN'S RALLY SET IN CITY

Dr. Kate S. Zerfoss, Nashville, Tenn., a graduate of the Tulane University School of Medicine, presided at the midyear board meeting of the American Medical Women's Association, December 3-4, at the Jung hotel. Internationally known women physicians throughout the United States were guests of the New Orleans branch.

Attending were Dr. Esther P. Lovejoy, first woman to serve as president of the International Association of Medical Women, who is widely known in the profession for her work in Europe during World War I. Dr. Catherine MacFarlane, noted for her work in cancer prevention research, also attended.

The program included a luncheon at the Jung hotel December 3 followed by a banquet at the Orleans Club; luncheon at Corrine Dunbar's and dinner at International House December 4. Dr. Elizabeth Bass, chairman of the New Orleans branch, was hostess.

### ANNOUNCEMENT OF VAN METER PRIZE AWARD

The American Association for the Study of Goiter again offers the Van Meter Prize Award of three hundred dollars and two honorable mentions for the best essays submitted concerning original work on problems related to the thyroid gland. The Award will be made at the annual meeting of the Association which will be held in Chicago, Illinois in April or May 1946, providing essays of sufficient merit are presented in competition.

### INFORMATION WANTED

The State Society has been requested to ask all members released from the armed forces to send the following information to the Directory Department of the American Medical Association, Chicago 10, Illinois:

Full name.

Date military service began and terminated.

Present address of residence and office, or a permanent home address to which mail may be directed.

Whether in practice, retired, or not in practice (on terminal leave, etc.).

If serving a residency in a hospital, indicate the date service is to begin and terminate.

Former permanent address if it is not the same as that indicated as present address.

### INFECTIOUS DISEASES IN LOUISIANA

The weekly morbidity report for the State of Louisiana for the week ending October 6 contained a statement of number of venereal diseases for the previous month. There were reported in this period 2040 cases of gonorrhea, 1508 of syphilis, 30 of lymphopathia venereum, 26 of chancroid, and 24 of granuloma inguinale. Report for this same week included the following diseases in numbers greater than ten: Unclassified pneumonia 125, cancer 124, pulmonary tuberculosis 52, septic sore throat 36, rheumatic fever 32, pneumococcic pneumonia 1, dengue 24, bacillary dysentery 21, typhus fever 14, and malaria 13. Three cases of meningococcus meningitis were reported and six cases of poliomyelitis, scattered throughout the state. There were also reported two cases of undulant fever; one from East Baton Rouge Parish and one from Pointe Coupee. Of the 13 cases of

malaria reported ten were contracted within Continental U. S. and three outside. For the week ending October 13 there were 30 cases of typhus fever reported, 18 of septic sore throat, 16 of pulmonary tuberculosis and 10 of scarlet fever. There was only one case of meningococcus meningitis and two cases of undulant fever reported this week; the former occurring in Orleans Parish and the latter in East Baton Rouge and East Carroll Parishes. Of the six cases of malaria reported, four were contracted outside of Continental U. S. and place of contraction was not stated for two. The report for October 20 showed a considerable increase over the previous week in the number of pulmonary tuberculosis cases, there being 43 reported. Also reported this week were 24 cases of mumps and 24 of septic sore throat, 16 of scarlet fever, 15 of diphtheria, 12 of hookworm, and 10 each of poliomyelitis and unclassified pneumonia. The poliomyelitis cases occurred in Caddo, De Soto, Orleans, Ouachita, Sabine, St. Mary and Tensas Parishes; Caddo having four and the other parishes one each. Two cases of malaria were contracted within Continental U. S., two outside and place of contraction of one was not stated. For the week which closed October 27, 42 cases of malaria were reported followed by 35 of septic sore throat, 34 of scarlet fever, 32 of diphtheria, 16 of pulmonary tuberculosis, 12 of typhus fever, and 10 each of hookworm and influenza. Rapides and Vermilion Parishes each reported one case of meningococcus meningitis, East Baton Rouge one of undulant fever, and poliomyelitis was reported by four parishes; Allen one, Caddo five, Orleans one and Sabine two. Of the 42 cases of malaria reported this week 28 were contracted within Continental U. S. and 14 outside of Continental U. S.

## HEALTH IN NEW ORLEANS

The Bureau of the Census, Department of Commerce, reported that during the week ending October 13 there were 123 deaths listed in the City of New Orleans of which 77 were white and 46 were colored, including a total of 10 under one year of age; 6 of whom were white. There was an increase of 34 deaths reported for the week which closed October 20. In the total of 157 there were 96 white and 61 colored. Also, during that week there occurred 19 deaths in children under one year of age; 10 white and 9 colored. The report of October 27 showed the number of deaths to be 76 white and 51 colored totaling 127. In this number only 1 was a white child under one year of age and 7 were colored. For the week closing November 3, 152 deathes occurred in this city; 97 were white and 55 non-white, including 13 infants which were mostly colored.

## THEODORE JOHN DIMITRY

Dr. Theodore John Dimitry, an active member of the State Society for many years, died in New Orleans on October 27. Dr. Dimitry practiced in New Orleans, specializing in ophthalmology, since his graduation from Tulane in 1901. He was chief of the Department of Ophthalmology of Louisiana State University and chief of the eye clinc at Charity Hospital.

## WOMAN'S AUXILIARY

Following are the officers of the Woman's Auxiliary to the Louisiana State Medical Society for 1945-46:

*Officers*

President—Mrs. Paul G. Lacroix, 3132 State Street Drive, New Orleans.

President-elect—Mrs. Arthur Long, 1367 Steele Drive, Baton Rouge.

First Vice-President—Mrs. Carroll Gelbke, 44 Willow Drive, Gretna.

Second Vice-President—Mrs. O. B. Owens, 1931 Military Highway, Pineville.

Third Vice-President—Mrs. B. C. Garrett, 4700 Fairfield Ave., Shreveport.

Fourth Vice-President—Mrs. A. G. McHenry, 1810 Riverside Drive, Monroe.

Treasurer—Mrs. Richard Vincent, 1441 Eleonore Street, New Orleans.

Recording Secretary—Mrs. Frank Jones, 810 Park Blvd., Baton Rouge.

Corresponding Secretary—Mrs. Willard Wirth, 402 Vincent Avenue, New Orleans.

*Chairmen of Standing Committees*

Archives—Mrs. Ansel Caine, 5301 St. Charles Avenue, New Orleans.

Bulletin—Mrs. Earl Clayton, Norco.

Cancer Control—Mrs. Herman B. Gessner, 119 Audubon Blvd., New Orleans.

Doctor's Day—Mrs. Geo. J. Taquino, 18 Fontainbleau Drive, New Orleans.

Finance—Mrs. Donovan C. Browne, 4920 St. Charles Ave., New Orleans.

Historian—Mrs. John S. Dunn, 8410 Pontchartrain Blvd., New Orleans.

Hygeia—Mrs. Leo. J. Kerne, Thibodaux.

Indigent Widow's Fund—Mrs. Aynaud Hebert, 2013 Napoleon Ave., New Orleans.

Legislation—Mrs. C. Grenes Cole, 4938 St. Charles Ave., New Orleans.

Organization—Mrs. Arthur Long, 1367 Steele Drive, Baton Rouge.

Parliamentarian—Mrs. A. A. Herold 1116 Louisiana Ave., Shreveport.

Press and ublicity—Mrs. Roy Carl Young, Covington.

Printing—Mrs. Daniel J. Murphy, 127 South Solomon Street, New Orleans.

Program—Mrs. J. E. Heard, 512 McCormick Street, Shreveport.

Public Relations—Mrs. Rhodes Spedale, 701 Eden Street, Plaquemine.

Red Cross—Mrs. William Kohlmann, 1330 Elenore Street, New Orleans.

Revision of By-Laws—Mrs. J. Kelly Stone, 72 Fontainbleau Drive, New Orleans.

War Participation—Mrs. Clarence Erickson, 423 Herndon Ave., Shreveport.

*Councilors*

First District—Mrs. Jules Myron Davidson, 100 Fontainbleau Drive, New Orleans.

Second District—Mrs. Roy B. Harrison 2327 Napoleon Ave., New Orleans.

Third District—Mrs. Jules E. Dupuy, New Iberia.

Fourth District—Mrs. William J. Sandidge, 522 Stephenson Street, Shreveport.

Fifth District—Mrs. DeWitt T. Milam, 1704 Island Drive, Monroe.

Sixth District—Mrs. Marquis C. Wiginton, Hammond.

Seventh District—Mrs. Olin Moss, 2011 Shell Beach Drive, Lake Charles.

Eighth District—Mrs. Hiram O. Barker, Horseshoe Drive, Alexandria.

*Advisory Board*

Dr. W. R. Metz, Chairman, New Orleans; Dr. King Rand, Alexandria; Dr. Charles Spedale, Plaquemine.

### MR. C. P. LITER ADDRESSES MEET

The Woman's auxiliary of the East Baton Rouge Parish Medical society met at the home of Mrs. M. L. Levy November 9. The social hour was followed with a business meeting, at which Mrs. J. O. Hoth presided in the absence of Mrs. Roy Rabelais.

Mr. C. P. Liter, the guest speaker, was introduced by Mrs. Edward J. Cailleteau. He addressed the group as a lay representative of the Blue Cross Hospital plan and explained the plan to the group. There are 13,600 people in Baton Rouge covered by the plan, 6000 of which are subscribers and 7600 dependents, he said. Hospitalization on the Blue Cross plan could be provided for 860,000 people for what it costs to operate the New Orleans Charity hospital, he stated, mentioning that 18,000,000 people are covered in the United States.

Co-hostesses were Mrs. John L. Beven, Mrs. Oswald Cosby, Mrs. F. U. Darby, Mrs. Wiley A. Dial, Mrs. J. P. Griffon.

Mrs. Charles Allen, Jr., Mrs. Felix Boizelle, Mrs. William Cook, Mrs. James R. Godfrey, Mrs. Robert Hansen, Mrs. Henry Hatcher, Mrs. Frank Jones, Mrs. Marian Landess, Mrs. Arthur D. Long, Mrs. Cecil Lorio, Mrs. Lionel Lorio, Mrs. C. S. McConnell, Mrs. E. E. Merse, Mrs. McHugh Simmons, Mrs. Richard Stephenson, Mrs. J. Eugene Toups, Mrs. W. A. Vennard, Mrs. Charles H. Voss and Mrs. M. A. Walker.

## TRANSACTIONS OF ORLEANS PARISH MEDICAL SOCIETY

### CALENDAR OF MEETINGS

December 17. Hotel Dieu Staff, 8 p. m.

December 18. Charity Hospital Medical Staff, 8 p. m.

December 19. The Orleans Society of X-Ray Technicians, 7:30 p. m.
Charity Hospital Surgical Staff, 8 p. m.
Clinico-pathologic Conference, Marine Hospital, 7:30 p. m.

December 20. Clinico-pathologic Conference, Touro Infirmary, 12 noon.
French Hospital Staff, 8 p. m.

December 21. I. C. R. R. Hospital Staff, 12:30 p. m.

December 27. DePaul Sanitarium Staff, 8 p. m.

December 28. L. S. U. Faculty Club, 8 p. m.
New Orleans Hospital Dispensary for Women and Children Staff, 8 p. m.

January 2. Mercy Hospital Staff, 8 p. m.
Clinico-pathologic Conference, Marine Hospital, 7:30 p. m.

January 3. Clinico-pathologic Conference, Touro Infirmary, 12 noon.

January 4. Medical Advisory Committee, Charity Hospital Visiting Staff, 11 a. m.

January 7. Board of Directors, Orleans Parish Medical Society, 8 p. m.

January 8. Eye, Ear, Nose and Throat Staff, 8 p. m.

January 9. Woman's Auxiliary, Orleans Club, 3 p. m.
Touro Infirmary Staff, 8 p. m.

January 14. Installation Meeting, Orleans Parish Medical Society, Jung Hotel, 7:30 p. m.

### NEWS ITEMS

The November scientific meeting, which was

held on the 12th, was as follows. How the Low Mortality Rate in Battle Casualties Was Accomplished in the Third United States Army, by Dr. Charles B. Odom. Penetrating Abdominal Injuries, by Dr. John F. Oakley. Medical and Surgical Experiences in Prisoner of War Camps in the Philippines and Japan, by Lt. Col. Edwin S. Kagy, M. C., U. S. A.

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Dr. Eugene H. Countiss was recently appointed by the president, in accordance with the by-laws of the Society, as librarian of the Society to fill the vacancy created by Dr. John R. Schenken's removal to Omaha.

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Dr. Wm. B. Clark has returned from Yepocapa, Guatemala, where he spent six weeks instituting a research project in the cause of blindness in patients infected with onchocerciasis in Guatemala. This project is sponsored jointly by the Caribbean sector of the Pan American Sanitary Bureau and the Department of Health of Guatemala. Dr. Clark will return to Guatemala in January to close out or continue the project, which is being continued in his absence, as the results of the investigation justify.

## MILITARY SERVICE

The following physicians have been released from active military service, and have resumed practice:

Robert E. Rougelot, 820 Maison Blanche Bldg.; Anthony J. Italiano, 725 Audubon Bldg.; L. S. Charbonnet, Jr., 706 Maison Blanche Bldg.; Edwin R. Guidry, 1343 Annunciation St.; W. Robyn Hardy, 637 Canal Bldg.; John S. Herring, 1521 Delachaise St.; Charles B. Odom, 1521 Delachaise St.; Cosmo J. Tardo, 725 Audubon Bldg.; J. O. Weilbaecher, Jr., 626 Canal Bldg.; Joseph A. Sabatier, Jr., 810 Hibernia Bldg.; Malcolm P. Schwarzenbach, 226 Verret St.; Pascal L. Danna, 946 Canal Bldg.; Morris Shushan, 1113 Pere Marquette Bldg.; Zachary J. Romeo, James S. Webb, 1207 Richards Bldg.; Harry Meyer, 301 Medical Arts Bldg.; Joseph D. Landry; Warren L. Rosen, 502 Medical Arts Bldg.; Clarence A. Bishop and Richard E. Selser, Charity Hospital; Horace L. Wolf, Touro Infirmary; Conrad G. Collins and Mims Gage, Tulane Medical School.

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Dr. Leon J. Menville was appointed to the advisory committee for the celebration of the 50th anniversary of the discovery of x-rays. November 5-10 was set aside for the observance of this anniversary; and the committee made plans for a nationwide educational program to acquaint the public with the development of the x-ray.

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Dr. John H. Musser was recently elected district vice-president, representing the Gulf states, of the Association of Pennsylvania Clubs, University of Pennsylvania alumni.

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Dr. George W. McCoy recently assisted the Chicago Board of Health in its scientific research work on poliomyelitis. Following this he attended the meeting of the National Foundation of Infantile Paralysis held in New York City.

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Drs. Emil Palik and John R. Schenken recently completed a paper entitled "Disseminated Granuloma Venereum," which is to be published soon in the American Journal of Clinical Pathology. This paper was presented by Dr. Schenken at the annual meeting of the American Society of Clinical Pathology.

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Dr. Alton Ochsner attended a meeting of the American Cancer Society recently held in New York.

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Dr. Neal Owens was recently elected first vice-president of the American Society of Plastic and Reconstructive Surgery.

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Dr. B. B. Weinstein recently addressed the Obstetrical and Gynecological Society of Mexico City. Dr. Weinstein also visited the Military school of medicine and the University of Mexico school of medicine while in Mexico City.

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Dr. Julius Lane Wilson spoke on the life of Dr. Trudeau at the 97th anniversary meeting of the Trudeau Memorial Guild.

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Dr. David R. Womack and Dr. Jack E. Strange were recently elected to office in the Ernie Pyle Post of the American Legion. Dr. Womack was elected commander and Dr. Strange was elected chaplain.

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## REMOVALS

Dr. Joseph N. Ané from 1201 Maison Blanche Bldg., to 604 Pere Marquette Bldg.

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Dr. Wm. K. Gauthier from 637 Canal Bldg., to 2049 Metairie Road.

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Dr. Gretchen Squires has removed to Florida; she assumed her new position as director of the Pathology and Clinical Laboratory of the Pensacola Hospital, Pensacola, on October 20. Dr. Squires will also maintain a full time private practice in clinical pathology.

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Dr. John R. Schenken has removed to Nebraska; he is director of Laboratories at the Methodist Hospital, Omaha.

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At the regular meeting of the Society held

Monday, November 12, 1945, the following nominations for Officers of the Orleans Parish Medical Society for 1945, and Delegates to the Louisiana State Medical Society for 1946-1947, were handed to the Secretary in accordance with the By-Laws:

President-Elect—Dr. H. Ashton Thomas
Endorsed by: Drs. Wm. H. Roeling and Frederick F. Boyce.

First Vice-President—Dr. Edmund M. Connely
Endorsed by: Drs. E. A. Ficklen, Edwin H. Lawson and Chas. Cox.

Second Vice-President—Dr. C. J. Brown
Endorsed by: Drs. Frank Chetta and H. Ashton Thomas.

Third Vice-President—Dr. Edgar Hull
Endorsed by: Drs. C. Grenes Cole and L. A. Fortier.

Secretary—Dr. Max M. Green
Endorsed by: Drs. C. J. Brown and E. L. Leckert.

Treasurer—Dr. Boni J. DeLaureal
Endorsed by: Drs. Randolph Lyons, M. D. Steiner and Walter Levy.

Librarian—Dr. Eugene H. Countiss
Endorsed by: Drs. Paul B. Lastrapes and Wilkes A. Knolle.

Additional Members

Board of Directors—Dr. E. L. Leckert
Endorsed by: Drs. Roy B. Harrison, J. E. Brierre and O. J. Burger.

Dr. J. O. Weilbaecher, Jr.
Endorsed by: Drs. O. C. Cassegrain and George J. Taquino.

DELEGATES TO THE LOUISIANA STATE MEDICAL SOCIETY

Dr. H. B. Alsobrook—Endorsed by: Drs. M. P. Boebinger and John T. Sanders.

Dr. Geo. C. Battalora—Endorsed by: Drs. Louis A. Monte and Wm. M. Roeling.

Dr. C. J. Brown—Endorsed by: Drs. Edwin L. Zander and Max M. Green.

Dr. Donovan C. Browne—Endorsed by: Drs. Louis Leggio and Max M. Greenberg.

Dr. W. R. Buffington—Endorsed by: Drs. Chas. Cox and John T. Sanders.

Dr. Frank Chetta—Endorsed by: Drs. C. J. Brown and L. S. Charbonnet, Jr.

Dr. Wm. B. Clark—Endorsed by: Drs. Paul B. Lastrapes and Wilkes A. Knolle.

Dr. Lucien A. Fortier—Endorsed by: Drs. Philip A. Boudreaux and J. C. Menendez.

Dr. Aynaud F. Hebert—Endorsed by: Drs. Louis Leggio and Max M. Green.

Dr. Sam Hobson—Endorsed by Drs. H. R. Unsworth and M. P. Boebinger.

Dr. Theo F. Kirn—Endorsed by: Drs. P. A. Phillips and John J. Irwin.

Dr. Edwin H. Lawson—Endorsed by: Drs. H. R. Unsworth and Chas. Cox.

Dr. E. L. Leckert—Endorsed by: Drs. Leon Menville, W. H. Roeling and L. A. Monte.

Dr. Jos. P. Palermo—Endorsed by: Drs. L. A. Fortier, J. M. Ciaravella and Spencer McNair.

Dr. E. J. Richard—Endorsed by: Drs. N. K. Edrington, A. E. Fossier and Spencer McNair.

Dr. Wm. H. Roeling—Endorsed by: Drs. J. E. Brierre and Percy A. Phillips.

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# BOOK REVIEWS

*Pulmonary Edema and Inflammation:* An Analysis of Processes Involved in the Formation and Removal of Pulmonary Transudates and Exudates, by Cecil K. Drinker, M. D., D. Sc., Cambridge, Mass. Harvard University Press, 1945, pp. 106. Price, $2.50.

This monograph consists of the Nathalie Gray Bernard Lectures given by Dr. Drinker at the Bowman Gray School of Medicine in December 1944 with an added chapter on artificial respiration. By the use of a new experimental technic to measure the lymph drainage from the lungs and a drug, thiourea, which causes pulmonary edema and pleural transudates, a considerable contribution has been made to our knowledge of the formation and disposal of fluid in the lungs. In the light of this new experimental work the author has written both readably and profoundly about the relations between lung structure and edema, the physiologic factors involved and the preventive and therapeutic measures available. There is little or nothing about inflammation as distinct from edema. The fundamental importance of anoxia in setting up a vicious cycle of pulmonary edema is clearly demonstrated. The section on artificial respiration is valuable because of the author's association with the development of the respirator bearing his brother's name and because of his own long experience with the comparative study of methods of resuscitation. This small book is stimulating reading. It should be of value to internists, thoracic and industrial surgeons, anesthetists and all others who are interested in pulmonary and car-

diac disease. There are both a carefully selected bibliography and an index.

JULIUS L. WILSON, M. D.

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*The Physiology of the Newborn Infant:* By Clement A. Smith, M. D. Springfield, Illinois, Charles C. Thomas, 1945. Pp. 312. Price, $5.50.

This eminently useful handbook of information by Dr. C. A. Smith, Professor of Pediatrics at Wayne University College of Medicine accomplishes very nicely the purpose of summarizing pertinent fundamental knowledge of physiologic peculiarities and characteristics of the fetus and newborn. The extensive bibliography following each section is well chosen; omission of some recent publications from an otherwise rather complete list seems unfortunate but is probably accounted for by wartime delays in publication. There are thirteen chapters, each sensibly divided into sections, ending always with a clinical smmary which crystallizes information applicable to situations commonly encountered in practice. Particularly outstanding are the chapters dealing with respiration and circulation, relating the application of cumulative research in these fields to rational methods of resuscitation.

The text is very well organized and follows an unusually excellent and readable pattern. It is well supplied with suitable charts, tables, and pictures and can be heartily recommended as a valuable handy source of information for all physicians having responsibility for the care of premature and normal newborn infants.

R. V. PLATOU, M. D.

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*Men Under Stress:* By Roy L. Grinker, Lt. Col. M. C., and John P. Spiegel, Major M. C., Army Air Forces. Philadelphia, Blakiston, 1945. Pp. 484. Price, $6.00.

With the exception of those who are too somber or fact-bound most persons will find in this excellent book a tremendous lot of valuable neuropsychiatric information to single it out for especial attention.

Under sufficient stress any individual may exhibit failure of adaption evidenced by neurotic symptoms. War, which of necessity is largely destructive, fortunately has some scientific and humane by-products. It stimulates intensive studies of man's complex somatic and emotional processes and teaches lessons which have useful applications in civilian life regarding the methods by which men adapt themselves to all forms of stress either in war or in peace. Therefore, the problem resolves itseld not in the detection of normality or its lack but in a study of the psychological methods of dealing with the harsh realities of human existence.

*Men Under Stress* tells what the men are like before they go into battle, thereby giving a cross-section of American youth. It describes combat fear and the many other kinds of neurotic disturbances—cases of changed personality, men who are "ghosted by lost comrades," men who have become hostile and aggressive. Sixty-five complete case histories are reported in detail. In this treatise are explained the various corrective treatments, including methods in which drugs have been used in psychiatric treatment of casualties of this war with unusual success and expediency.

Doctors Grinker and Spiegel have taken infinite pains to produce an astonishingly remarkable work which your reviewer unhesitatingly designates as a medical classic.

C. P. MAY, M. D.

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*Personal Mental Hygiene:* By Don Thomas Verner Moore, M. D., O. S. B., Ph. D. New York, Grune & Stratton, 1944. Pp. 331. Price, $4.00.

This book is unusual in that it combines orthodox psychiatry and religious truths. Such a combination will have to take place sooner or later and it is much needed already. To deal effectively with the personality such concepts as anger, hatred, love, charity, greed and pride must be used and used in such a way that they carry their long accepted connotations. New knowlege only supplements old truths. New psychiatry can only supplement established moral values, at least in the wisest application of psychiatry.

This is by no means a textbook of psychiatry and such important subjects as anxiety are treated very superficially. Psychopathic personality and schizophrenia are handled effectively by using the personalities of Goldsmith and Swinburne as illustrations. The use of famous people as examples of personality problems is a valuable part of this book. The emphasis on a healthy family circle is well taken in this book. This subject is of the greatest importance since serious functional psychoses rarely occur in harmonious homes. The author's estimation of the causes of the distintegration of family life must certainly be difficult for modern Americans to accept, but I believe that psychiatric experience will bear him out. The book is full of references to great religious teachers of the past as well as to psychiatric and non-psychiatric literature. He also makes use of his knowledge of neuro-physiology and pharmacology in describing the handling of various cases, which he describes in sufficient detail to make the book well balanced.

WALKER THOMPSON, M. D.

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*The Bacterial Cell:* By René J. Dubos, with an Addendum by C. F. Robinow. Cambridge, Mass., Harvard University Press, 1945. Pp. 460. Price, $5.00.

Dr. Dubos has here developed the substance of his eight Lowell Lectures, delivered in Boston in 1944, into a comprehensive and fascinating account

of the newer knowledge concerning the physical and biochemical structures of bacteria, as well as their genetic and physiologic behavior. The subject has been treated with special emphasis on its relation to bacterial virulence, immunity and chemotherapy, so that it is a valuable reference for the person interested in medical bacteriology or the larger problems of infectious disease, whether he be investigator, teacher or student. The treatment of the material is thorough and abundantly documented by references, the splendid bibliography being one of the most attractive features of the book. At the same time this is much more than a mere dull compendium, as Dr. Dubos has analyzed the available data in terms of what is yet unknown and frequently throws forth challenging ideas or questions which should serve as stimulation and guide to future research.

Dr. Robinow's chapter on nuclear apparatus and cell structure of rod-shaped bacteria is also noteworthy since it presents overwhelmingly convincing evidence, much of it photographic, of the existence and activity of nuclear material arranged in discrete units such as chromosomes and behaving like similar entities in other organisms. Many bacteriologists have been inclined to regard with considerable reserve, if not downright skepticism, the earlier claims for the demonstration of nuclear structures in bacteria; to these persons, Dr. Robinow's reproductions should be an eye- and mind-opener.

It is not intended to detract from the commendation of this book when one notes the existence of occasional minor errors, chiefly typographical, which are no doubt due to the difficulties of preparing such a work for publication under the exigencies of war-time. This reviewer believes that *The Bacterial Cell* should and will have not only wide distribution but also a profound influence on bacteriological thinking.

MORRIS F. SHAFFER, PH. D.

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*Preventive Medicine:* By Mark F. Boyd, M. D., C. P. H., 7th ed. rev. Philadelphia and London, W. B. Saunders Company, 1945. Illus., pp. 591. Price, $5.50.

This seventh edition of a widely accepted text in the fields of preventive medicine and public health preserves the format of the previous editions. While the same thirty-seven chapters occur, many of them with headings identical with those of the sixth edition, many have been largely rewritten to present the newer information on the subjects concerned. New plates, tables and charts are used, while some of the older ones which have trade-marked the book have been omitted. The bibliography is up to date and is more inclusive than previous ones. No typographic errors have been detected.

In short this is an adequate revision of a good text in preventive medicine in the moderate price range.

RDLPH H. HEEREN, M. D.

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*Virus as Organism:* By Frank M. Burnet, M. D., F. R. S. Cambridge, Mass., Harvard University Press, 1945. Pp. 134. Price, $2.00.

This is one of the well worth while publications of the Edward K. Dunham Lectures, put out by the Harvard University Press. Burnet, of Australia, needs no introduction as an authority on the viruses. His studies of viruses and the viral diseases are so well known that the author's name and his accomplishments are well known to the medical profession.

In this monograph Burnet discusses the virus diseases from a consistently biologic angle. He states that it is impossible not to look upon the viruses as living organisms which will reproduce and survive changes in environment as do other organisms. Illustrative of the fact that virus diseases breed true to type is the example of mumps which was discovered by Hippocrates 2500 years ago and which has been transmitted from individual to individual for twenty-five centuries without change in so far as the type of disease that it produces is concerned.

The first three chapters of the monograph have to do with the general studies of viruses that have been made in the past few years. Parenthetically it might be remarked that it has only been in the last 15 years that the medical profession has come to appreciate the importance of the viruses as disease producers and to study intensively their characteristics both in the laboratory and in man. The succeeding chapters each deal with one of the common and important viral disorders—herpes simplex, poliomyelitis, psittacosis, smallpox, yellow fever and influenza. There then follows a short chapter in which the author draws some conclusions from the material he has presented in the preceding chapters.

This book is intended primarily not for the bacteriologist, the virologist and the laboratory scientist, but rather for the practitioner of medicine. It summarizes reasonably succinctly the information that is available about the viruses; it discusses the more important viral diseases and more particularly their causative virus and the evolutionary and ecological aspects of the specific virus responsible for the disease. The book is truly most fascinating. It is written with restraint and good sense; it presents material with which the average doctor is unacquainted and about which he should know more than he probably does.

J. H. MUSSER, M. D.

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*The Fundamentals of Electrociographic Interpretation:* By J. Bailey Carter, M. D., F. A. C. P. Springfield, Ill., Chas. C. Thomas, 1945. Pp. 406, illus. Price, $5.00.

In the second edition of this book there has been some rearrangement of the subject matter and some additions made. The bibliography follows each chapter instead of being at the end of the book. Most of the reproductions are good. The description of some are not complete. The reviewer feels that all the abnormalities in the electrocardiogram should be pointed out as well as the ones under discussion. There would also be a difference of opinion in the interpretation of some of the curves, i. e. Figure 46 page 91 is said to show myocardial damage evidenced by slurring of QRS 1—slurring and notching of QRS 3. One may have good grounds for claiming that this curve is within normal limits. The slurring of QRS 1 is very slight and QRS 3 has the lowest amplitude of the three leads. A little fuller and more up-to-date description of the Wolff Parkinson and White syndrone would be of help to the beginner.

There are several curves showing typical left and right bundle branch block. He labels them simply bundle branch block. The reviewer feels that the type of curve in Figue 99, page 148, should be called left bundle branch block. The one shown in Figures 106 & 107 on Page 152 should be labeled right bundle branch block. Chest leads in these types of curves when they are not typical help to place them under their proper heading.

The author with some justification seems to think that the interest in the chest leads has detracted from the proper study and appreciation and importance of the llmb leads. However, it must be remembered that in some cases the chest leads will reveal the presence of myocardial damage while the limb leads are equivocal. The author relies principally on what he terms simplified technic in the recording of chest leads — described as follows. "After the three standard leads are recorded, the left arm electrode is removed and applied to the chest in the fifth interspace in the midclavicular line; lead wire connections remaining undisturbed. The electrode is held in place by the fingers of the patient. Lead I. & III. on the control board are selected in turn. These are called Lead IV. & Lead V. or Lead CR 4 and inverted CF4". The reviewer prefers to place the chest electrodes in the C1—C2—C3—C4—C5 and C6 position using the so-called central terminal or the left leg as the distant electrode. This will not be necessary of course in all cases but where the chest leads are considered necessary it is best to place the electrode on more than one point. This seems to be in accord with most of the writers on this subject. It is important to avoid as many sources of confusion as possible. The author feels that serial curves are of much greater assistance than the chest leads. Some might think that this is a little over-emphasizing; however, there is no question about the great importance of serial curves.

Chapter 22 Concepts of Coronary Disease in the space of ten pages is brought out the principal points of the accepted view of cardiologists of today.

Chapters 35 and 36 — Graphic Findings in Various Diseases and Clinical Value of Electrocardiography contains much that will be helpful to the beginner.

In the case histories the electrocardiogram is shown and described under each case which is an improvement over the first edition.

The beginner will find much in this book that will help him.

J. M. BAMBER, M. D.

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*Neuro-Ophthalmology*: By Donald J. Lyle, B. S., M. D., F. A. C. S. Springfield, Ill., Chas. C. Thomas Co., 1945. Pp. 395. Price $10.50.

Ophthalmology is indebted to the author for an outstanding contribution. In four hundred pages the subject is covered in a practical and reasonably complete manner. Difficult and complex concepts are expressed in words of almost one syllable.

Nineteen chapters, a bibliography of almost eighteen hundred references, two hundred and thirty-four illustrations, a comprehensive table of contents and an index make up this interesting, instructive and timely volume. The section on Intracerebral and Orbital Neoplasms is of special interest because accurate localization is usually very difficult for the Ophthalmologist. Ocular reflexes and associated structures of the mid-brain is another subject which is presented in an exceptionally understandable manner. It is made as simple as such a complex subject can be made. The eye manifestations of head injuries will be of special interest to ophthalmologists and others espe-

cially in the military service. Although presented in but fifteen pages, the fundamentals are given and their applications illustrated with exceptional clarity. The illustrations are ample in number and excellent in quality. They really illustrate.

No ophthalmologist will make a mistake in reading and re-reading this outstanding volume.

CHAS. A. BAHN, M. D.

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## PUBLICATIONS RECEIVED

American Medical Association: Council on Pharmacy and Chemistry Reports 1944. New and Non-official Remedies—1945.

American Red Cross, Washington, D. C.: American Red Cross First-Aid Textbook.

The Commonwealth Fund, New York: Government in Public Health, by Harry S. Mustard, M. D., LL. D.

Paul B. Hoeber, Inc., New York: General and Plastic Surgery, by J. Eastman Sheehan, M. D.

Lea & Febiger, Philadelphia, Pa.: Modern Urology for Nurses, by Shelia Maureen Dwyer, R. N., B. S. and George W. Fish, M. D.

Charles C. Thomas, Springfield, Ill.: Prescribing Occupational Therapy, by Wm. R. Dunton, Jr., M. D.

The Williams & Wilkins Co., Baltimore, Md.: The Basis of Clinical Neurology, by Samuel Brock, M. D.